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JANUARY 1967 / Vol. 87, No. 1 / Pages 1 - 32

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CONTENTS

H. D. Lees Series: Institutional Environmental Health and Safety: The Minnesota Story 1
G. S. Michaelsen

Severe Myxedema Following Inadvertent Removal of an Ectopic Thyroid Resembling a Thyroglossal Duct Cyst 7
John H. Saiki, M.D.

Prolonged Allergic Reaction to Oxacillin 10
Gustavus A. Peters, M.D., Edward D. Henderson, M.D., and Joseph E. Geraci, M.D.

The Development and Perpetuation of Obesity 13
Nancy L. Wilson, B.S., Seymour M. Farber, M.D., L. S. Kimbrough, M.D., and Roger H. L. Wilson, M.D.

Ascorbic Acid and Carbohydrate Metabolism II: Effect of Supervised Sucrose Drinks upon Two-Hour Postprandial Blood Glucose in Terms of Vitamin C State 18
A. T. S. H. Setyaadmadja, M.D., E. Cheraskin, M.D., D.M.D., and W. M. Ringsdorf, Jr., D.M.D.

Section on Pain and Distress: Worthy of Attention 22
John S. Lundy, M.D.

The Effect of LSD on the Phantom Limb Phenomenon 22
S. Kuromaru, M.D., S. Okada, M.D., M. Hanada, M.D., Y. Kasahara, M.D., and K. Sakamoto, M.D.

Book Reviews on Pain 28

Gaylord W. Anderson, M.D. 30
Stewart C. Thomson, M.D.

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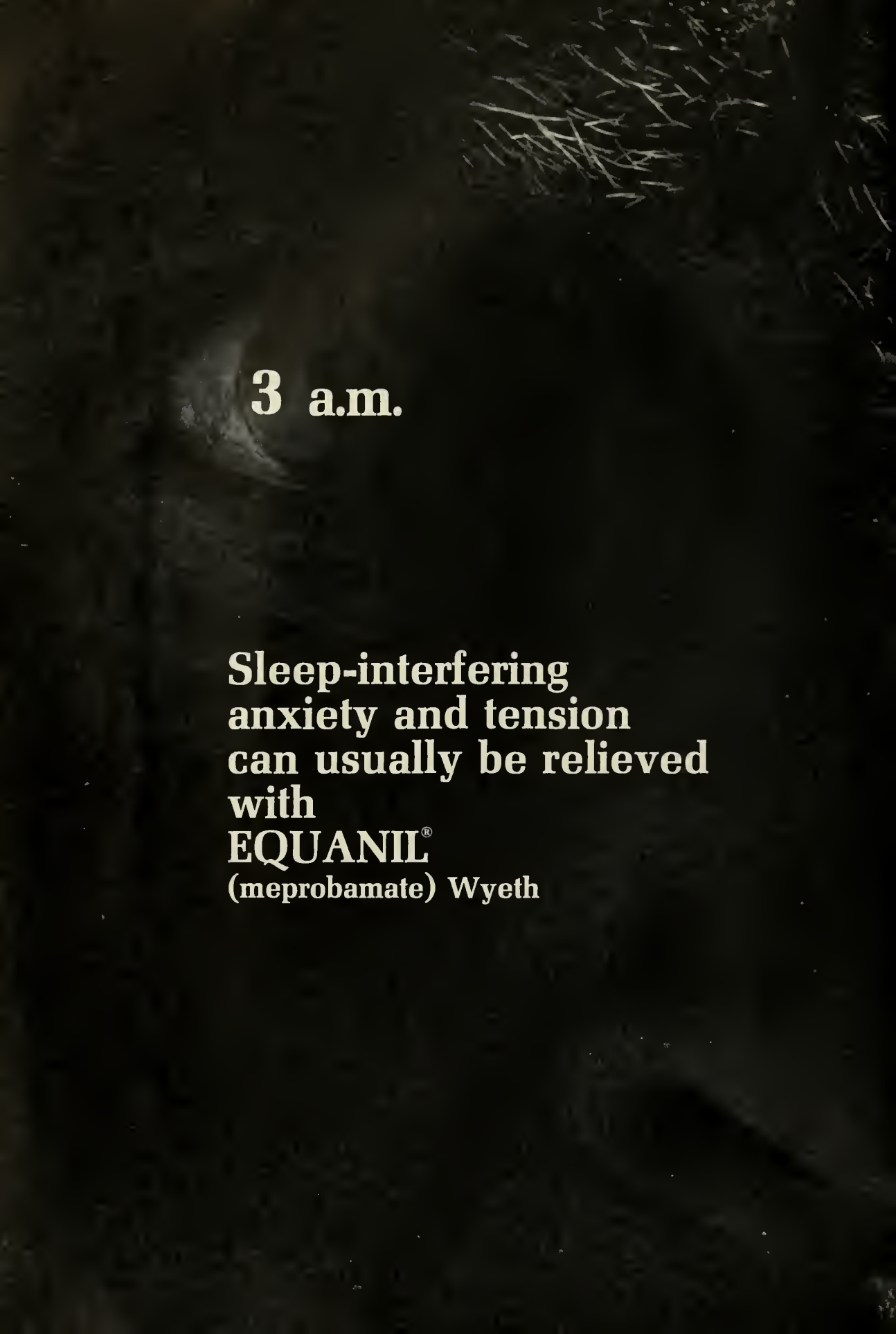
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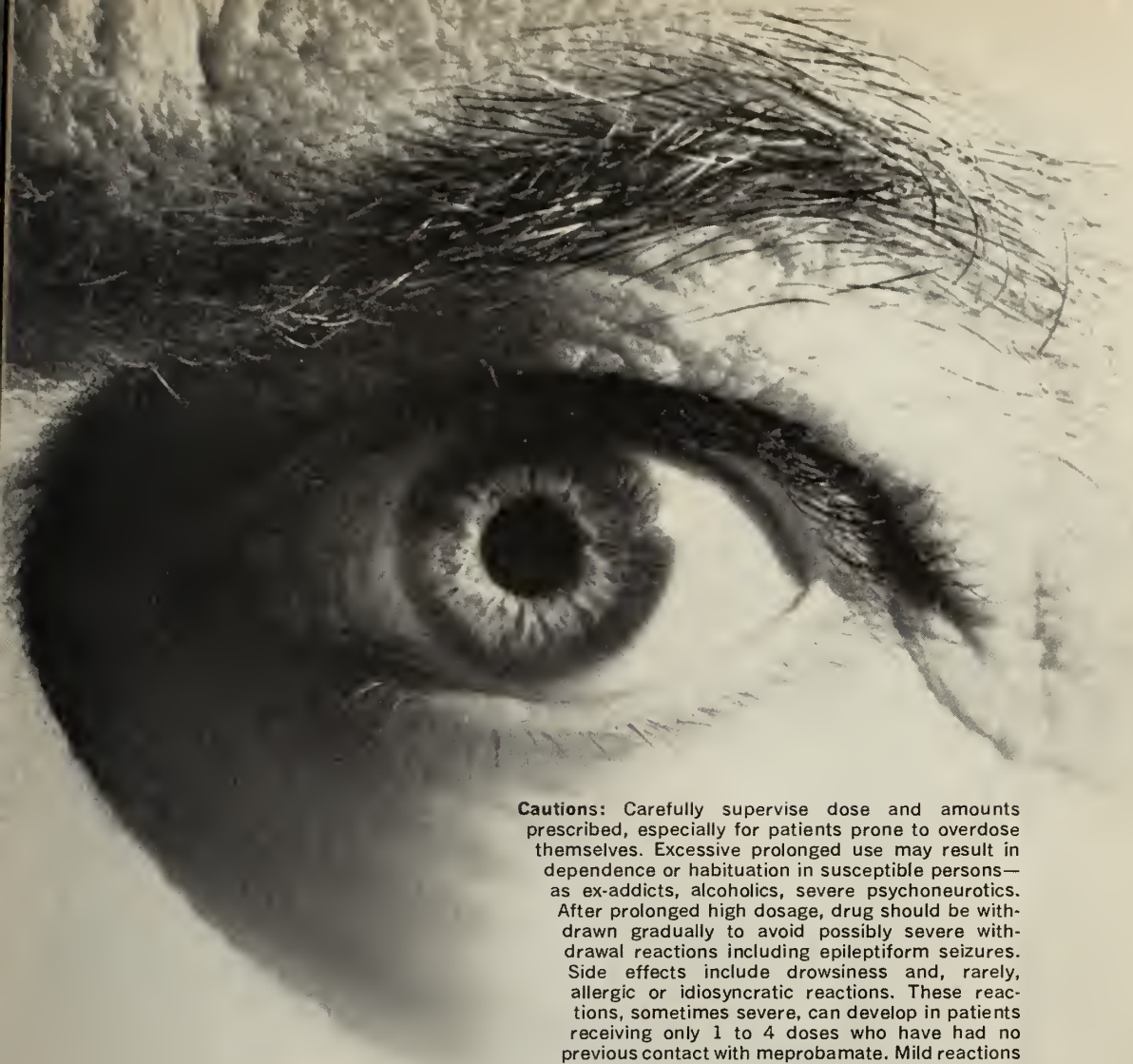
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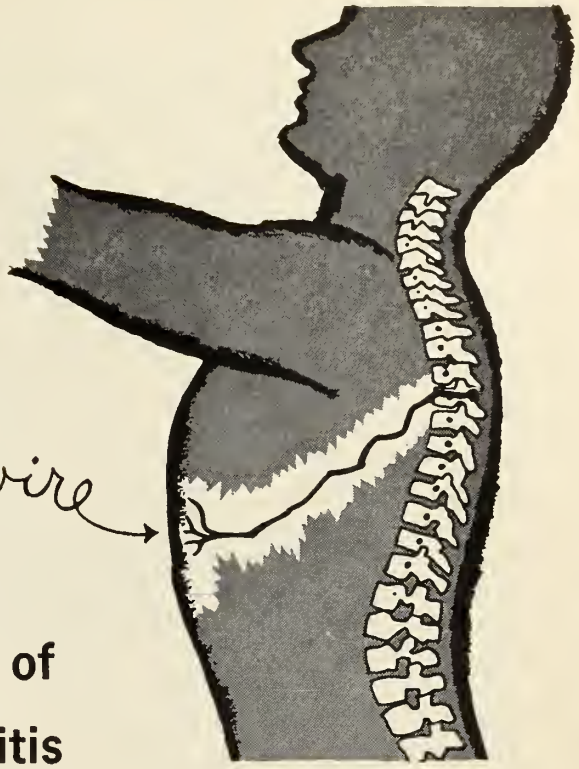
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References: (1) Baker, A. G.: Penn. Med. J. *63*:697-698 (May) 1960. (2) Smith, R. T.: New York Med. *8*:16-19 (Aug. 20) 1952. (3) Smith, R. T.: Med. Clin. N. America (March) 1957. (4) Lehrer, H. W.; Lehrer, H. G., and Lehrer, D. R.: Northw. Med. *54*:1249-1252 (Nov.) 1955. (5) Sforzolini, G. S.: Arch Ophthal. *62*:381-385 (Sept.) 1959. (6) Shupala, E.: W. Virginia Med. J. *56*:191-193 (June) 1960. (7) Xander, G. W.: Western Med. *1*:14-17 (Sept.) 1960. (8) Combs, F. C., and Canizares, O.: New York J. Med. (Mar. 15) 1952, pp. 706-708. (9) Marsh, W. C.: U.S. Armed Forces Med. J. *1*:1045 (Sept.) 1950. (10) Love, T. R.: Rocky Mountain Med. J. *50*:873 (Nov.) 1953.

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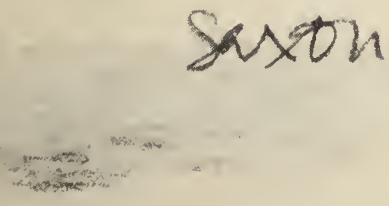
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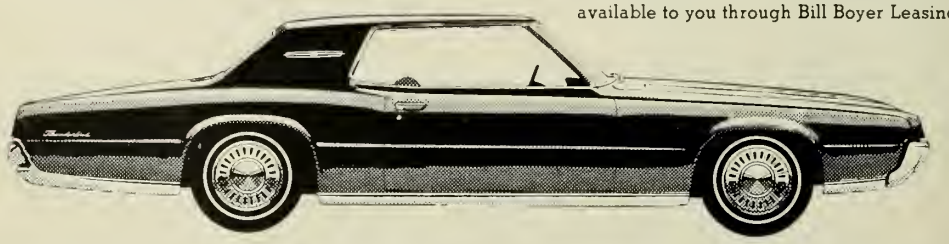
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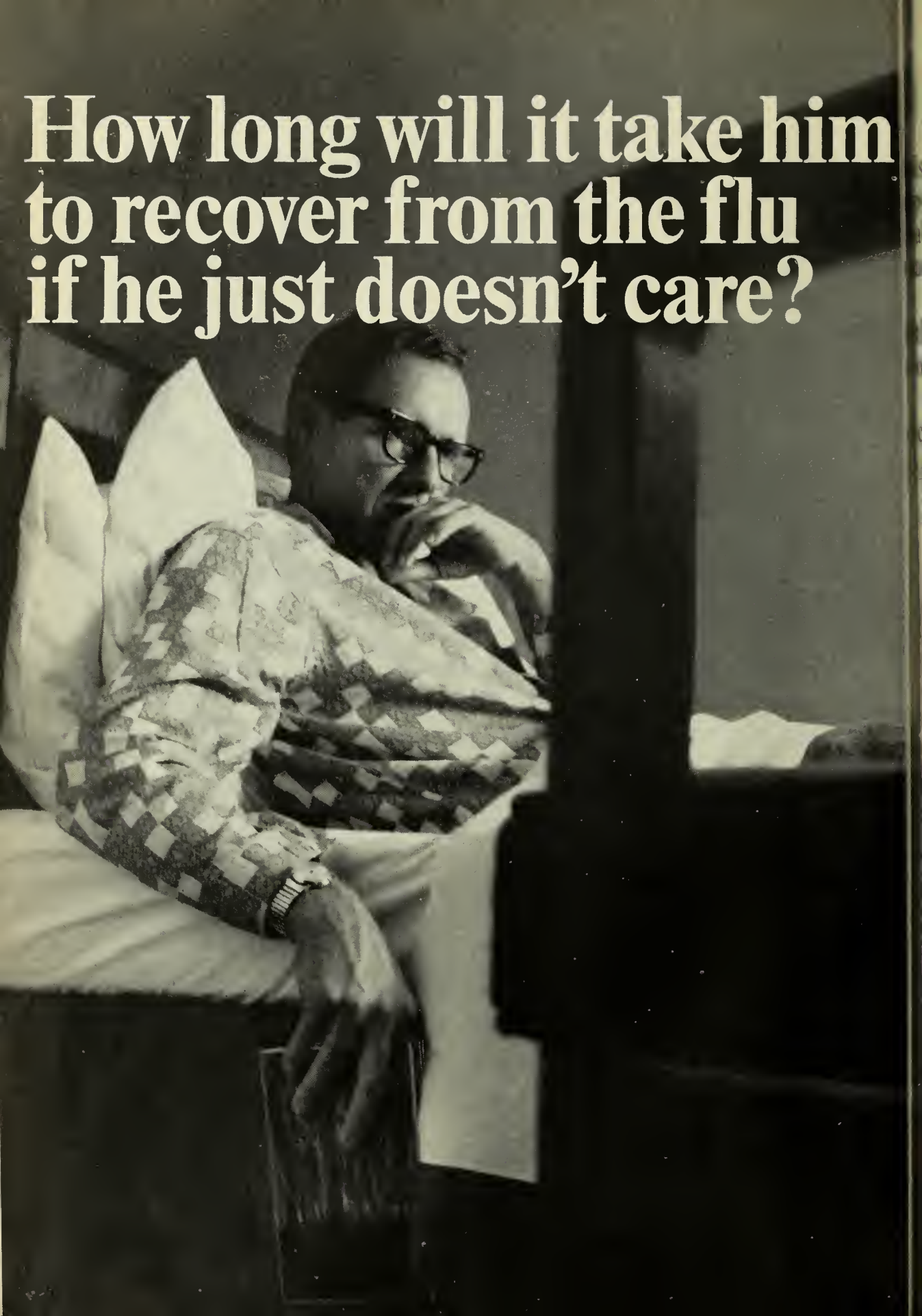
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H. D. LEES *Series*

Institutional Environmental Health and Safety: The Minnesota Story

G. S. MICHAELSEN

Minneapolis

THE UNIVERSITY OF MINNESOTA Health Service has had an environmental health and safety program for thirty years, although it has not always operated a program as complete as this title would imply. In 1934 Harald Graning, now an assistant surgeon general of the U.S. Public Health Service and director of the Division of Hospitals and Medical Facilities, was appointed as a sanitary inspector. His chief responsibilities were related to the swimming pools and water supply. A group known as Minnesota Dames made a survey of married student housing in 1938. This served to draw the attention of the administration to the problems of healthy and safe housing for all students. The health service made a rather complete study of the situation and in 1940 the administration requested the health service to take on the task of housing inspection for all students, in addition to the sanitary inspections already being made. This was done by part-time housing inspectors under the supervision of a part-time public health engineer.

As the sanitary inspections and housing program increased in volume and scope, it became apparent that some full-time personnel would be needed. In 1949 Mr. Richard Bond was employed as the first full-time public health engineer to administer the environmental health and safety program. With the assistance of a number of part-time people, the scope of the program enlarged to include more work on food services, water supply, and waste disposal.

In order to strengthen the administration of the program, a sanitary code was prepared for and adopted by the board of regents in 1951. It then became university-wide and top level administrative policy that there was to be concern for environmental health and safety programs on the campus. With this administrative recognition and concern, more requests for assistance on health and safety matters were received and, as a result, in 1953 a sanitarian and an industrial health engineer were added to the staff. About this same time the health service was asked by the administration to concern itself with industrial-type accident hazards and radiation hazards control. The industrial health engineer, with the assistance of a full-time X-ray technician, initiated the health physics program. He also began laying the groundwork for the safety program.

By 1954 the increased use of radioisotopes and the installation of increased numbers of ionizing radiation producing pieces of equipment required the appointment of a full-time qualified health physicist. Upon the recommendation of

G. S. MICHAELSEN *is director, Division of Environmental Health and Safety, University Health Service, University of Minnesota, Minneapolis.*

the All-University Advisory Committee on Safety, a full-time safety supervisor was added to the staff in 1955.

The volume of work associated with the environmental health and safety program, resulting from increased student enrollment and academic staff, as well as increased requests for assistance, necessitated adding a second health physicist and second sanitarian in 1956. In 1958 the third health physicist and a third sanitarian were hired.

In 1961 the sanitary code was amended to reflect more accurately the additions and changes in the environmental health and safety program. The activities have continued to grow so that in 1962 a second safety engineer was added to the staff, and in 1963 a fourth sanitarian was employed. As of this writing, the environmental health and safety professional personnel consist of 1 public health engineer, 1 industrial health engineer (director), 2 safety engineers, 1 safety technician, 3 health physicists, and 5 sanitarians. In addition to the participation in the environmental health and safety program of the health service, these people have teaching duties in the school of public health. Some of them are also actively engaged in research.

The environmental health and safety program is divided into 5 functional areas: (1) public health engineering, (2) occupational health, (3) safety, (4) radiological health, and (5) general sanitation. The engineering portion of the program concerns itself with water supply, sewage and waste disposal, and plumbing defects. In addition to several municipal water supply systems serving university facilities, there are about 37 well water supplies owned and operated by the university. Sewage disposal installations at university facilities vary from discharging raw sewage into the Mississippi River to discharging into municipal systems. In between there are privies, septic tanks, and imhoff tanks. All of these situations need to be evaluated from a public health standpoint. It is necessary to make surveys of the plumbing installations in university buildings, looking particularly for cross-connections and other plumbing defects which might endanger the quality of the water supply. This is particularly troublesome in laboratory buildings.

The occupational health portion of the program is concerned with problems of overexposure to toxic dusts, fumes, gases, vapors and mists, inadequate ventilation, inadequate illumination, noise, and exposures to extremes of temperature. These are problems which one usually associates with industry, but within a complex university all the occupational health hazards which one finds in industry can be found, although the numbers exposed to any one material or condition are likely to be less than one would encounter in industry. Examples of the types of problems which have been worked on include exposures to lead, a wide variety of solvents, mercury, noise, carbon monoxide, asphyxiation, insecticides and welding fumes, and a large variety of ventilation problems.

SAFETY

As the division name might suggest, the safety efforts are being directed toward improvement and control of the environment in such a manner that the exposures of faculty, civil service staff, students, and the public to accidental injury will be reduced to a minimum. The majority of the work and activities are of an engineering nature. Educational activities, as such, are limited and have as their purpose instruction in control of the environment or recognition of hazards rather than safety education per se. The responsibilities for safety education are clearly set forth in the president's letter on university safety policy and become the responsibility of the faculty or civil service supervisor.

The scope of the safety program includes industrial, research, instructional, athletic and recreational activities performed by students, faculty and staff on the various campuses, and stations. It includes the safety of the public while visiting the campuses or attending public gatherings in university buildings. It concerns itself with fire as it affects the life safety of any of the previously mentioned groups. The program does not include traffic safety in any of its aspects. The program is concerned with the activities of outside contractors only to the extent that their activities may affect the safety of faculty, students, staff, and the visiting public.

Initial activities were directed toward inspection of the physical facilities of the university. These efforts served the purpose of familiarizing personnel with the physical facilities and activities. However, the size of the physical plant, the varied and often changing activities of the departments and the student and faculty population, made a safety program based on these procedures impractical. Although no longer a major activity of the safety program, some routine inspections of university buildings are made. Many of these inspections have resulted from conditions noted at the time of an accident investigation or while checking a request regarding a specific safety problem. Physical inspection of particularly hazardous building occu-

pancies, such as the chemistry building, chemical engineering building, and ag-biochemistry building, is made. The various laboratories operated in conjuction with the college of medieal seiences have also been subject to periodie inspections. Upon completion of sueh inspections, it is the policy to submit written recommendations for correetion of hazards noted.

In the carly stages of the safety program an aceident reporting system was developed which has been continued and provides information as to student, employee, and public injuries. From such records information can be developed as to areas producing unusual exposures and actual individual accidents of a nature that require investigation can be rapidly considered.

A summary of the accident reeords for the academic year 1964-65 shows that employee injuries occurred as follows: service departments 34% of the total, University Hospitals 34%, academic department 26%, and out-state stations 3%. The frequency rate for the entire university was 5.41 and the severity rate was 140. This compares with the national average for all industry of a frequency rate of 6.12 and a severity rate of 682. A major source of faculty and staff injuries was handling materials, with the hands being the part of the body most frequently injured. Student injury experience indicates that a slightly higher percentage of injuries occur off campus than on, that the relationship of injuries to male and female is in proportion to the male and female enrollment, and that the injuries serious enough to require hospitalization bear a elose relationship to the total number of injuries. On-campus injuries occurring to students indieate that there is about an even division between athletic injuries and all others. The athletic injuries indicate that intramural aetivities create the greatest number of accidents, but have a very large participation in comparison with other athletic activities. Of the nonathletic injuries, laboratory accidents and accidents to pedestrians are the leading sources of injury. During the period covered there were 168 disabling injuries, of which 75 required hospitalization for a period of 240 total days. The average hospital stay for on-campus hospitalized injuries was three and one-half days.

At the present time the safety program is essentially operating on a request basis. By this it is meant that assistance on eontrol of accidents is primarily handled on the basis of receiving requests from various personnel or departments regarding recognized safety programs. It is felt that the majority of the personnel desire to have a safe working environment and, if somehow advised of unsafe conditions that may be associated with their activities, they will take what action is economieally possible to incorporate the necessary safety features in the environment. Unfortunately, many of the personnel either do not reeognize unsafe conditions or beeome so involved in their specific activities that they do not have time to devote their attention to a comprehensive evaluation of the problem. It is the practice of the safety engineers to periodically visit the various divisions and departments which may have recurring safety problems and, by their presence, initiate recall of items which need discussion. This approach is particularly productive in the plant services department, purchasing department, and with university services.

There are several other approaches now being used to create an interest or eall attention to hazards. One sueh effort is the publication of a health and safety bulletin used to outline specific hazards associated with known University activities. To date, there have been 21 such bulletins published, covering subjects on carbon tetraehloride, fire extinguishers, dicyclohexycarpodimide, laboratory accidents, hazards of material being left on roofs, flammable liquids, safety cans, machine guarding manual, duplicators, plastic wastepaper baskets, protective clothing and equipment, safe atmospheres, compressed gas cylinders, automatie alarms, combustible metals, walk-in cold rooms, extension cords, paraffin, stepstools, waste chemieals, and emergency signs.

A second method is the publishing of safety standards which serve as a guide in the purchase, use, storage, and disposal of known hazardous items. To date, 2 such standards have been published, the first on compressed gas eylinders and the second on extension cords. A third method, now being initiated to alert personnel to specific hazards, is the publication of special hazard bulletins, of which 1 has been published. This bulletin is on perchloric acid and outlines case histories of past accidents, as well as setting forth proper safety measures for the handling of this particular chemieal. Still another method of gaining interest of staff and research personnel is the holding of group meetings in the various departments, whieh will last from one-half to one hour. Here a very informal discussion of safety problems directly related to the activities of the department is carried on.

In some departments and divisions, safety committees or single individuals have been appointed whose duties are to take particular interest in the safe environment of the department or division and to advise the safety engineers of

any unusual activities, hazards, or conditions which may develop within their area.

Because of the importance of incorporating safety into new buildings and new activities, the safety engineers, acting as part of the team of the Division of Environmental Health and Safety, are able to review plans for new buildings and for major remodeling of existing structures. Suggestions and recommendations are forwarded to the advisory architect for the university. The division receives copies of minutes from the university machine shop committee and from room assignments, and is given the privilege of reviewing the grants and contracts which have been submitted to the board of regents for approval. The division is also represented on the All-University Advisory Committee on Space Allocation. These are all helpful in discovering activities which need investigation as to hazard control.

The authority for specific actions deemed necessary in the prevention of accidental injuries originates from the board of regents approved environmental health and safety code. All safety activities are advisory in nature and police action is not a part of the program. Occasionally, authority for suggested corrections is requested by others and the code provides such a reference. The president annually appoints an All-University Safety Committee, whose purpose it is to evaluate the safety needs of the university and to act in an advisory capacity to the safety engineer. Until last year, this committee's activities were directed primarily toward industrial safety. However, at the suggestion of the Division of Environmental Health and Safety, the name was changed, a student representative appointed, and the interests of this committee expanded to include safety on a university-wide basis.

HEALTH PHYSICS

Health physics, radiological health, or radiation safety may be defined as the protection of people from the damaging effects of ionizing radiation. Although this is a simple definition, the program which is necessary to achieve that objective is quite complicated.

The sources of ionizing radiation which are amenable to evaluation and control at the university consist of a large number and variety of radioactive isotopes, diagnostic and therapeutic X-ray installations, X-ray diffraction units, electron microscopes, Van de Graaff generators, and a linear accelerator. Isotopes are being used by 167 AEC licensees. Of these, 117 are in the college of medical sciences, 26 in other departments on the Minneapolis Campus, 16 on the St. Paul Campus and 8 in Austin, Cloquet, and Duluth. The radiation-producing machines consist of 42 diagnostic X-ray machines, 20 dental X-ray units, 14 X-ray diffraction units, 10 X-ray machines for general research purposes, 6 therapy X-ray machines, 4 electron microscopes, 1 linear accelerator, and 1 Van de Graaff generator. These are located in 24 different departments.

One of the major elements of the program in protecting people from the damaging effects of ionizing radiation is the monitoring of personnel to determine just what their exposure amounts to. This is accomplished by the use of film badges. It is the practice to have the people wear these badges for two weeks. The films are then exchanged and the worn film is sent to the film badge company for development and interpretation. The data is returned from the company in the form of an IBM data sheet. These are reviewed by the health physicist to see if there are any high exposure levels which should be investigated to determine the cause and to institute corrective measures if this should be warranted. There are over 500 people now being monitored in this way. It is the decision of the health physicist as to whether any particular isotope worker is to wear a film badge, since the film badge is not suitable for all types of exposure. The Atomic Energy Commission Regulations require, however, that anyone who may likely receive 1/4 of the quarterly permissible dose shall be provided with a film badge. The records of the exposures are kept on file and become a part of the permanent record of the exposure of these individuals during the time they are employed by the university and their work involves exposure to ionizing radiation. This has particular value in any litigation which might arise concerning possible overexposure to ionizing radiation. The film badge program is sufficiently flexible so that special monitoring procedures can be carried out as needed.

Currently, when an individual is supplied with a film badge, his name is given to the staff examining unit of the health service and he is requested to come in for a radiation medical examination. This serves to establish the person's physical condition before any possible radiation exposure, as a protection against future unjustified claims of radiation injury. It also provides an opportunity to eliminate those from radiation work who, for medical reasons, should not work around sources of ionizing radiation.

Since a complete evaluation of personnel exposure cannot be arrived at by film badges alone, it is necessary to conduct area surveys. This covers a multitude of services which are provided

by the health physicists at the site of the use of radioactive materials or radiation-producing machines. It is necessary to make periodic surveys for radiation and radioactive contamination in all areas where ionizing-producing machines and materials are used. There are some areas where more highly radiotoxic materials are used or where the potential for exposure is greater and, consequently, these areas are under surveillance more frequently than areas where the radiation hazard potential is less.

The area survey includes a review of the nature of the project with the principal investigator in order that the health physicist may have a complete understanding of the work which is to be done. In this way he is able to discuss and evaluate the health physics aspects of the project. Discussions are also held with all the members of the group working with the isotopes, so they will have a complete understanding of the health physics precautions to be observed. This becomes a form of in-service training. The storage of radioisotopes in the laboratory, proper labeling of the laboratory, as well as proper shielding, disposal of radioactive wastes, need for film badges, and general techniques in the handling of radioisotopes are discussed.

During the course of the project, visits are made to the laboratory from time to time to review the techniques employed, pick up radioactive waste, and make complete surveys to determine whether the laboratory facilities have become contaminated. Air samples are collected in the laboratory periodically if there is any possibility of air-borne radioactivity resulting from the operations in the laboratory. When a project involving the use of radioactive materials is terminated, it is necessary to survey the laboratory to see that it is free of contamination and to remove, for disposal, any radioactive waste which remains.

The health service has equipment on hand which can be used in case of an emergency to decontaminate laboratories and even handle major spills of radioactive materials.

As a part of exercising some element of control over the use of radioactive isotopes on the campuses of the university, all requisitions for the purchase of such materials must be approved by the health physicist before the order will be placed. In addition, all shipments of radioactive materials (with a few exceptions) are delivered to the health service. These shipments are logged in, examined for possible damage and delivered to the purchaser. A record is kept of the approval of the requisition, the receipt of the shipment and the delivery of the material to the user. When there is a particularly troublesome procedure in removing the radioactive material from the shipping container to the laboratory equipment, the health physicists provide supervision and monitoring.

One of the most time-consuming and troublesome aspects of the health physics program is the matter of waste collection and disposal. At the outset of the program it was deemed desirable and virtually necessary for the health service to provide a waste disposal service in order to insure the proper disposal of radioactive wastes. This program has grown from the collection of radioactive materials from a few laboratories to a very extensive program which requires many hours of service each week to make it effective.

Three basic methods are used for waste collection and disposal:

1. Dilution in the sewage flow

Some radioactive materials may be disposed of in the sewerage system if the levels of activity are low and the half-life of the material is less than thirty days. This disposal can be made by the investigator, or he can place the waste in suitable containers, properly labeled, and it will be disposed of by the health physicists. In either case, it is necessary that the health physicist be advised as to the amount and nature of the radioactive material. Where this method of disposal is used in a particular laboratory, the trap of the sink is provided with a radioactive warning tag to alert the plumbers to a possible radiation hazard.

2. Incineration

Research with radioisotopes includes the injection of these materials into animals. To make certain that these animals are not disposed of in violation of the AEC regulations, the health service has obtained an AEC license to incinerate contaminated animals. These animals are incinerated on a special hearth in the large university incinerator. The ash in the incinerator is monitored and records are kept to see that all this is done within the requirements of the AEC license.

3. Radioactive waste storage

Radioactive solid and liquid wastes which are combustible and noncombustible, and which cannot be disposed of by methods 1 and 2, also accumulate. Special containers for the liquid wastes are available and the laboratories are requested to use these. When these are full, the health service picks them up and replaces them with empty containers. When a sufficient quantity accumulates, they are transported to the

Rosemount Storage Facility where an outdoor evaporator has been constructed. The liquid wastes are poured into the evaporator and the accumulated solid residue is placed in containers for storage and ultimate off-site disposal.

For the solid wastes 2 containers are available, 1 for combustible and 1 for noncombustible materials. These are properly designated G.I. cans and the laboratories are required to obtain these. The health service provides plastic liners for the cans. When they are full, the health physicists remove the plastic liners and place them in fiber drums. As they accumulate, they are transported to the Rosemount Storage Facility for ultimate off-site disposal.

Health physics supervision of the nuclear engineering laboratory in the department of chemical engineering is also a part of the health service program. The laboratory consists of a nuclear reactor simulator, graphite natural uranium exponential pile, and the gamma irradiation facility.

GENERAL SANITATION

The general sanitation program is divided into 5 major areas of concern: (1) housing, (2) food, (3) swimming pools, (4) water and sewage, and (5) insect and rodent control.

The major housing activities are associated with off-campus facilities such as sororities, fraternities, cooperatives, religious foundations, rooming houses, apartments, and private homes. It is university policy that students under 21, not married and not living with blood relatives, must live in housing approved by the university. The housing bureau, under the dean of students, administers this program and policy, and the health service establishes the standards of the healthful housing and makes the housing inspections. Recommendations for approval or disapproval are based on meeting or not meeting these standards. The health service makes inspections of rooming houses, apartments, and private homes at the request of the housing bureau and reports its findings, with recommendations as to approval or disapproval, to the housing bureau. The sororities, fraternities, cooperatives, and religious foundations are also under the general supervision of the dean of students. These are inspected routinely and approximately annually, and the results, with recommendations for disapproval when warranted, are sent to the dean of students.

The health service cooperates closely with the city health department in Minneapolis, St. Paul, and Duluth in this housing inspection program. Several hundred housing inspections are made each year.

The on-campus housing consists of dormitories and married student housing units. These are not inspected routinely, but only as requested by the director of university services and by the director of University Hospitals. Many such inspections are made each year, however.

In the area of food sanitation the present program consists of attempting to make an annual sanitary survey of each of the 30 food service facilities operated by the university. These consist of the dormitory food services, Coffman Union, Shevlin Hall, the Campus Club, the St. Paul Dining Center, the Hospital Food Services (including the infant formula room), the Kirby Food Center on the Duluth Campus, and the numerous concession stands at football, baseball, basketball, and hockey games.

Sanitary surveillance is provided for the 8 milk and food caterers with whom the university has contracts. Sanitary surveillance is also provided for special events such as the Campus Carnival, Foreign Student's Festival, and special banquets or picnics. There are about 200 food and drink vending machines on the Minneapolis and St. Paul campuses. The sanitary quality of the food and drinks dispensed from these machines is also under observation. The university-operated dairy plant and the food stores facility are also kept under sanitary observation.

The sanitarians of the division work closely with the health service physicians in the investigations of all suspected incidents of food or drink borne disease. The sanitarians also assist in the plan examination of food service facilities.

The present program regarding the sanitation of the swimming pools involves the weekly bacteriological, chemical, and physical examination of the 5 swimming pools on the Minneapolis and St. Paul campuses and also the pool at the president's residence. Daily operational reports are received from the pool operators. These are used mainly as checks against major errors in operation or as explanations for results obtained in the analyses.

The work on the water supplies, plumbing, and sewage disposal systems consists of weekly samples for bacteriological examination from the St. Paul campus supply, monthly samples from the Rosemount Research Center and yearly samples from the outlying stations. Supervision is given to the chlorination of new water mains, repaired mains, and extensive new services in buildings.

In the area of insect and rodent control, the health service acts in a consultative and advisory

(Continued on page 12)

Severe Myxedema Following Inadvertent Removal of an Ectopic Thyroid Resembling a Thyroglossal Duct Cyst

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Ectopic thyroid is commonly misdiagnosed as a thyroglossal duct cyst and removed with the unfortunate development of severe myxedema. Although this occurrence has been reported before, the author believes that this case is unique because this patient was without normal thyroid function from the age of 5 to 17. At this time he was cretinous in appearance, very strongly emphasizing the possible sequelae of a preventable condition and the need for establishing the accurate diagnosis.

CASE REPORT

A 17-year-old Navajo boy presented at our outpatient clinic in June 1964 complaining of a sore over his right parotid gland. This subsequently proved to be a furuncle, which healed with appropriate treatment. Review of his record revealed that in 1953 he was admitted for removal of a thyroglossal duct cyst just below the hyoid bone. Following surgery he had an uneventful recovery and was discharged. The specimen was reported histologically to be normal thyroid tissue. The patient was lost to follow-up until his present admission.

On physical examination his appearance was striking. He looked cretinous. His stature was that of a 10-year-old boy. Both his height and arm span were 55 in. The crown pubis length was 29 in. and the pubis to floor length was 26 in. His weight was 90 lb. His face was myxedematous and there was coarseness of scalp hair. There was a small scar just below the hyoid bone and no thyroid tissue was palpable. His skin was very dry and scaly. The skin over his elbows and knees was hyperkeratotic and showed the classically described "tree bark" appearance. He had no beard growth, no axillary hair, and only a few pubic hairs. His testes were smaller than an adult. Neurological examination revealed a classical myxedematous ankle jerk. His thinking and actions were slowed.

Routine hemoglobin, hematocrit, sedimentation rate, white cell count and differential white cell count, blood urea nitrogen, two-hour postprandial blood sugar, and urinalysis were normal. The serum cholesterol was 244 mg. percent. The protein-bound iodine was 0.4 μg. percent. His basal metabolic rate was minus 40. A serum carotene was 94 μg. percent. His 24-hour urine collection showed a 17-ketosteroid level of 2.4 mg. and a 17-hydroxycorticoid level of 3.1 mg. An electrocardiogram showed low voltage with flat P and T waves in all leads. X-ray of his chest was normal. X-ray of his hands and wrists revealed a bone age of 10 to 11 years.

Treatment with dessicated thyroid was begun. The initial dosage was ½ gr. daily and this was slowly increased by small increments over a three-month period to 4 gr. daily. His features have changed strikingly. Although his weight has remained 90 lb., he has grown 2 in. in height. The protein-bound iodine is now 5.2 μg. percent. His bone age three months after treatment was 12 to 13 years and nine months after therapy was 14 years.

DISCUSSION

In 1962 Quigley and associates[1] reviewed the Anglo-American literature on subhyoid median ectopic thyroid and found 9 cases reported. All were misdiagnosed as thyroglossal duct cysts before surgery and 5 during surgery. Five of the 9 subsequently developed severe myxedema.

Thyroglossal tract anomalies usually present before the age of 10 and occur more commonly in males. Ectopic thyroids are usually high and midline in front of the hyoid bone and no thyroid exists in the normal location.[2]

The incidence of ectopic thyroid is very small. In reviewing approximately 7,000 cases of thy-

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Fig. 1. Close-up view of face at the time of admission illustrating myxedematous facies



Fig. 2. Comparison of patient with a normal 17-year-old boy



Fig. 3. Dry scaly skin of patient on right

roid disease, Clute and Cattell[3] found 74 with thyroglossal tract anomalies. Of these, 2 were ectopic lingual thyroids. In 1940 Gross and Connerly[4] reported 198 cases of thyroglossal cysts and sinuses and found 1 ectopic thyroid. Of 105 cases of thyroglossal tract anomalies reported by Ward, Hendrick, and Chambers,[5] 6 had ectopic thyroid tissue and 1 developed myxedema. Nachlas[6] reviewed 128 cases and noted 1 case of ectopic thyroid. In 1962 Turcot[7] reported 80 cases of thyroglossal tract anomalies, 5 of which were ectopic thyroids.

Complications occur in only half of thyroglossal tract anomalies and are most commonly recurrent infection and cosmetic disfigurement. Carcinomatous change is rare.[8] Fish and Moore[9] in 1963 found 21 reported cases of cancer in lingual thyroid arrests and 13 cases of cancer in thyroglossal duct remnants.

The treatment of thyroglossal tract anomalies does not always require surgery. In 1961 Brown and Judd[10] emphasized that not all cysts and sinuses require surgery. However, when complications arise they pointed out that conservative surgical approach often leads to recurrence and urged that the original resection be done by the radical Sistrunk procedure with removal of the central part of the hyoid bone. To avoid removal of ectopic thyroid tissue, diagnosis before surgery by use of I^{131} has made recognition of ectopic thyroid simple in most cases.[11,12] To establish diagnosis during surgery, Gross and Connerly[4] recommended first that the tissue be biopsied and secondly that the presence of a normal thyroid gland in its normal position be ascertained. When a subhyoid median ectopic thyroid is found Quigley and his associates[1] recommend that the gland be split longitudinally in the midline and each half moved laterally to its normal position beneath the strap muscles. When this is not possible it has been recommended to transplant the ectopic thyroid.[13]



Fig. 4. Three months after onset of therapy

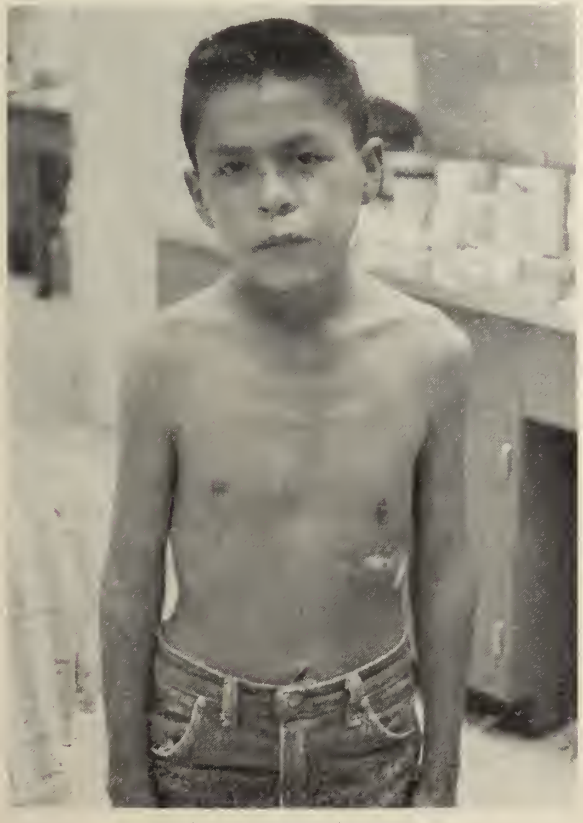


Fig. 5. Six months after therapy



Fig. 6. Nine months after therapy

SUMMARY

In summary, a case of severe myxedema resulting from inadvertant removal of an ectopic thyroid has been presented to emphasize the importance of establishing accurate diagnosis before removing a thyroglossal duct cyst.

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Prolonged Allergic Reaction to Oxacillin

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REPORTS OF ADVERSE REACTIONS to oxacillin are rare, but one of our patients, an elderly atopic physician, experienced an allergic reaction consisting of acute urticaria of the trunk and lower parts initially followed by delayed angioedema and swelling of the salivary glands, lips, and tongue, the latter symptoms recurring periodically for approximately four months.

Sodium oxacillin (5-methyl-3-phenyl-4-isoxazolyl penicillin sodium) is resistant to gastric acidity and to penicillinase. Geraci and associates[1] have shown it to be effective when given orally against penicillinase-producing staphylococci, and it is indicated for strains of staphylococci resistant to penicillin G.

Like other penicillins, it can produce hypersensitivity in some persons. Reactions to oxacillin are more likely to occur in those who already have had a reaction to some form of penicillin. The reactions have been infrequent and usually mild, consisting of minor gastrointestinal symptoms, skin rash, pruritus, or urticaria.[1,2] Moderate elevation of serum glutamic oxaloacetic transaminase (SGOT) has been observed in a few instances.[3] More recently, Freedman[4] has described suspected bone marrow suppression and hepatocellular dysfunction in an allergic woman who received 3 gm. of sodium oxacillin by mouth daily for approximately three months. Steroids and antihistamine drugs were used to promote recovery. Pas and Quinn[5] also reported on cholestatic hepatitis with elevated alkaline phosphatase, SGOT, and sulfobromophthalein findings following use of sodium oxacillin.

REPORT OF CASE

A 79-year-old retired physician was known to have had ragweed hay fever and sensitivity to fish and gum acacia, but he had not been aware of being sensitive to penicillin. His only known previous contact with penicillin had been an injection of 300,000 units of procaine penicillin G approximately twelve years previously for purulent bronchitis. He had become sensitized to gum acacia after testing it on himself and using it in the treatment of nephrosis.

On Nov. 2, 1964, the patient had fallen from a ladder, injuring his left leg and sustaining multiple bruises and skin lesions. The ecchymoses gradually cleared, but the skin lesions were slow in healing. By the end of November, small areas of induration and cellulitis were noted. Fever with tachycardia was present. On Nov. 25, orthopedic advice was sought.

Material for culture was taken from a lesion, and treatment with sodium oxacillin, 0.5 gm. four times a day, and hot packs was prescribed. After two days without improvement, the patient was hospitalized, and the use of oxacillin and hot wet dressings were continued.

On Dec. 1, the patient noted bluish urticarial, hemorrhagic skin lesions, from tiny size to 6 cm. in diameter, distributed diffusely over the lower trunk, the scrotum, and the legs. He also had nasal stuffiness, coryza, and epistaxis. His tongue was swollen and his mouth was sore, making eating uncomfortable. The administration of oxacillin was discontinued immediately.

The laboratory reported that the culture produced staphylococci sensitive to penicillin G (0.1 μg. per milliliter) and to erythromycin (1 μg. per milliliter). Also, Escherichia coli were found which were sensitive to streptomycin (5 μg. per milliliter), the tetracycline group (5 μg. per milliliter), and ampicillin (20 μg. per milliliter). The leukocytes numbered 13,900 per cubic millimeter of blood: 87% neutrophils, 8% lymphocytes, 4% monocytes, and 1% eosinophils. The platelet count, coagulation time, hemoglobin quantity, and prothrombin time were within normal limits. Roentgenologic study of the left leg showed vascular calcification and a bony plaque in the proximal part of the calf, but the bones were normal.

To ameliorate the allergic reaction the patient was given diphenhydramine hydrochloride (Benadryl®) orally, 50 mg. for the first dose and 25 mg. four times daily thereafter, plus calamine lotion for symptomatic relief of pruritus. On Dec. 2, the tongue was still swollen, but on Dec. 3, the urticarial and petechial lesions receded, with improvement each day until the patient was dismissed from the hospital Dec. 6. At no time did he have any asthma.

During the rest of December the open lesions were dressed with nitrofurazone (Furacin®)-impregnated gauze, and by Jan. 4, 1965, the cellulitis had subsided

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completely. On this same date, however, swelling of the tongue and enlargement of the submaxillary glands began to recur periodically. Nasal bleeding, nasal stuffiness, and diarrhea were present for several hours, but there was no recurrence of urticarial lesions. On Jan. 13, edema of the right side of the lower lip occurred and subsided gradually. During the night of Jan. 21, the tongue and submaxillary glands became edematous. On Jan. 25, edema affected the lower lip. On Feb. 6, the tongue began to swell late in the evening and edema of the submaxillary glands developed later, all symptoms disappearing by the following morning. On March 4, the mucous membrane of the mouth was slightly swollen and irritated. On March 12, there was swelling of the right half of the tongue, spreading gradually to the entire tongue and involving the submaxillary glands in about one and one-half hours and then the sublingual glands. During this episode epinephrine, 0.2 ml. in 1:1,000 solution, was given subcutaneously without much effect, although the swelling in the tongue and salivary glands disappeared in six to eight hours. The sublingual swelling persisted for twenty-four hours, and the soreness and tenderness in the mouth lasted about forty-eight hours.

A series of skin tests for sensitivity to penicillin were performed April 15, 1965, at which time the patient had been free of symptoms for about a month. First, penicilloyl polylysine (Cilligen®), in a solution containing 6×10^{-5} moles of Cilligen® per 0.14M of saline with 0.005M phosphate at pH 7.4, was injected intradermally to raise a bleb about 6 mm. in diameter. Within three minutes there was a reaction of grade 4 (on the basis of 0 to 4). This diminished to about grade 1 in the next ten minutes but flared an hour later to grade 3. Afterward, penicillin G, in a solution containing 1,000 units per milliliter, produced negative results on a scratch test using 1 drop and on intradermal injection of 0.05 ml. with a fifteen-minute period of observation for each test.

Oxacillin, 1.25 mg. per milliliter, produced negative results on a scratch test and an intradermal test. The patient had no systemic or localized reaction immediately after the skin testing but experienced swelling on the right side of the tongue about six days later.

On Nov. 23, 1965, repetition of the intradermal test with Cilligen® brought negative results.

COMMENT

Although only Cilligen® evoked a positive reaction among the skin tests, our clinical impression, based on the patient's urticaria and angioedema, was that he was probably sensitive to penicillin. Therefore, he was advised to avoid it and all its homologues in the future. It was not surprising that the test responses to penicillin G and oxacillin were negative, since in cases of urticarial reaction to penicillin—unless they are severe to the point of exfoliative dermatitis—the skin test response to penicillin G is often negative. The prompt reaction to the Cilligen® skin test was surprising but fits the present concept that in penicillin hypersensitivity the major antigenic determinant is the penicilloyl-protein conjugate. Levine and Ovary[6] have shown that antibodies in rabbits are formed especially well against the benzyl-penicilloyl-lysine grouping. Also, one may wonder whether the last episode of tongue swelling was related to the skin testing with penicillin G the week before.

Parker and associates[7] pointed out that the degradation products of penicillin, such as penicillenic acid and its penicilloyl derivative, readily form protein complexes or conjugates responsible for penicillin hypersensitivity. They also showed that the Cilligen® was not immunogenic, but yet it was capable of producing a wheal and flare in a high percentage of persons sensitive to penicillin. Budd and associates[8] reported that 20 of 22 patients with a history of clinical allergy to benzathine penicillin reacted positively to penicilloyl tests, whereas only 2 of 40 control subjects reacted thus. Penicillin G was found to be considerably less effective in detecting the hypersensitivity than was the Cilligen® antigen. Rytel and co-workers[9] showed that this haptene complex was able to produce positive skin reactions in 35% of persons with a history of penicillin allergy, and they believed that a strongly positive skin test response indicates a ninefold greater risk of systemic reaction to the administration of penicillin than does a negative response. Brown and associates[10] reported no life-threatening or anaphylactic reactions occurring in 13,350 patients with negative response to Cilligen® skin tests. VanArsdel[11] and Siegel and Levine[12] have recommended that solutions of penicillin G and of Cilligen® both be used in testing for sensitivity to penicillin, because the patient may react to one antigen and not to the other, and also because the acute anaphylactic reactor may not be reactive to the Cilligen® antigen. There is considerable immunochemical evidence that penicillins other than potassium penicillin G can introduce the penicilloyl determinant;[13,14] cross reactivity is likely to occur, since almost all of them form penicillenic acid.

SUMMARY

An acute urticarial allergic reaction occurred five days after initiation of oxacillin therapy in a patient not known to be sensitive to penicillin. Periodic reactions characterized by angioedema and glandular swelling developed about a month later and recurred intermittently for the next four months. The patient was an atopic physician with a history of ragweed hay fever and of sensitivity to fish and gum acacia. Skin testing produced only negative reactions to penicillin G and oxacillin but an immediate positive reaction (and seven months later, a negative reaction) to intradermal injection of penicilloyl polylysine.

Cilligen® supplied by Sigma Chemical Co., St. Louis

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Continued from page 6

INSTITUTIONAL ENVIRONMENTAL HEALTH AND SAFETY: The Minnesota Story—

G. S. Michaelsen

capacity; the actual control program is operated by the plant services department. The sanitarians investigate reports of insect and rodent infestation, give advice regarding control and, when needed, advise this department of infestations when they are found.

OUTLYING FACILITIES

For many years the program of the division was limited to service to the Minneapolis and St. Paul campuses. When the radiological health program was added to the division, there was also added direct budgetary support from the central administration. It was recognized that the program of the division was expanding and becoming more extensive, reaching into non-student related activities so that it was no longer proper to ask income from student fees to carry the entire budget. A few years later the division budget was increased in order to provide environmental health and safety services to all parts of the university, regardless of their location.

There are fifteen locations at which the university has some type of operation outside of the Minneapolis and St. Paul campuses. At the present time the Duluth campus and the Hormel Institute at Austin receive the most attention. At Duluth, service is given in the area of off-campus housing, occupational health and safety, radiological health, food service and the swimming pool. At the Hormel Institute radiological health and occupational health and safety receive most of the attention. Several visits a year are made to both facilities.

Visits to the other facilities are largely limited to annual visits, unless there are requests for additional service. There is considerable work of a general sanitation nature at the outlying facilities, for here there are about 36 well water supplies, 4 swimming pools and 1 natural bathing beach, 7 food services, and a wide variety of individual sewage disposal systems.

Even though the program just described is designed to meet in part the needs of a large educational and research institution, it should be recognized that problems of an environmental health and safety nature exist in institutions regardles of type or size.

As soon as a space with 4 walls, a ceiling, and a floor is provided and occupied with people, problems of an environmental health and safety nature arise. Matters related to comfort arise immediately—crowding, temperature control, humidity control, ventilation, lighting, toilet facilities and, right behind them, adequate means of egress in the event of an emergency, and problems of sanitation if food is to be prepared and consumed within this space.

So from this simple "institution" to the most complex one there are environmental health and safety problems and the number, variety, and severity increase with the complexity of the institution. Fortunately, it is not necessary to live with these problems in an uncontrolled fashion. They are all amenable to solution and control by the proper application of the knowledge and skills which are at hand.

The Development and Perpetuation of Obesity

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OBESITY IS ONE of the most important medical hazards in the United States today. It shortens the life-span, particularly increasing the incidence of significant cardiovascular disease, gallbladder disease, and diabetes. Many other common causes of death also occur at an earlier age in the obese than in the lean. This excess mortality is unnecessary since obesity is preventable.

Our present efforts toward early detection and prevention of crippling or killing disease make it timely to change our emphasis from treatment to prevention of obesity. Many factors leading to the development of obesity are known; most of these can be anticipated and preventive measures invoked. But after obesity has developed, treatment is much more difficult, since it is the nature of obesity to perpetuate itself. Although in an occasional case specific endocrine or other factors may be involved which would alter the therapeutic approach, the vast majority of patients, and even some of those with endocrine defects, fall into the categories discussed in this paper.

FAMILIAL AND AGE PATTERNS IN OBESITY

It is now possible to predict the likelihood of the development of obesity and the age at which this may occur. The earliest contributing factor, prenatal nutrition, may activate a genetic tendency toward overweight.[1] When one parent is overweight, there is a 50% chance that the child will be obese; this probability is increased to 80% when both parents are overweight.[2] This propensity is not seen in adopted children, even when adopted very young. Studies of adolescent girls reveal a familial and probably genetic predisposition to the development of obesity with normal food intake.[3] As Seltzer and Mayer suggest, "Presumably, the prime prerequisite is a physique with at least a moderate amount of endomorphy under normal nutritional conditions." Many middle-aged obese persons eat no more and often eat less than persons of normal weight of similar sex, age, and occupation. This reflects not only lessened physical activity but good energy conservation in the acts of sitting, lying, and standing which occupy 80% of their time, representing a "somatic trait" favoring obesity.[4] In general, endomorphy is a predisposition to the laying down of fat in individuals apparently consuming a normal diet for their age, sex, and height.

There are certain periods during life in which obesity is likely to develop: infancy, the time of starting school, puberty, and, in females, the first pregnancy and the menopause. In both sexes after the age of 25, an increment of 10 lb. with each decade has been so consistently found in "normal" adults that previous height and weight tables were adapted to show these as "normal" age changes. These have more recently been replaced with recommendations to maintain the best weight of the person at age 25.[5]

A pattern of obesity can be established in infancy. Overfeeding the infant leads to a fat baby who tends successively to become a plump child, an obese adolescent, and an overweight adult. Yet many parents, for sociological or psychological reasons, find a prime source of satisfaction and pride in their baby showing a more rapid growth and weight gain than other children. In the last fifty years American boys have become 6 to 8% taller but 12 to 15% heavier than their fathers, and maturation has occurred at an earlier age.[6] Concurrent with this has been

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the development of earlier atherosclerosis, especially of the coronary arteries.

A study of height and weight records of children in 3 public schools in Hagerstown from 1937 to 1939, followed up twenty years later when the average age was 31, indicated that overweight children tended to become overweight young adults.[7] Of overweight boys, 86% became overweight adults; of average weight boys, 42% showed a susceptibility only one-half as great to obesity in adulthood. This difference was even more striking among the girls, where the figures were 80% and 18%, respectively.

Unfortunately, much of our knowledge of nutrition and growth comes from experiments on laboratory animals which are uncertain in translation to man. The commercial raising of livestock gives emphasis to as large and rapid growth as possible with sacrifice of the young animal. Since this is not the goal in human nutrition, Youmans questions the benefit of forced growth and early maturation and considers the possibility that these may cause a shortened total life-span.

However, the incidence of overweight in the United States is increasing. At least 10 million teen-agers, representing 15 to 20% of the age group, are overweight.[8] The proportion is even higher in adults, since it includes those whose patterns of obesity continue, with an added group who become overweight after the age of 25. Thirty percent of all men and 40% of all women over 40 years of age are more than 20% above their best weight. Part of this can be attributed to the fairly constant food intake over a long period during which actual needs are steadily decreasing. A 10-calorie-per-day surplus becomes 3,650 calories in one year or an increase of about 1 lb. of body weight; in ten years this becomes the 10-lb. general trend in body weight increase for the population.[9]

PHYSICAL ACTIVITY AND CALORIC NEEDS

In this century, possibly the most salient factor in caloric imbalance contributing to increase of obesity at all ages is decrease in physical activity. The active use of human muscle power has been replaced by relative inactivity due to the increased development of controls operating other sources of power, but the maintenance of ancestral eating habits has resulted in a positive balance of calories. In obesity, relative inertia may be a cause or a consequence; both must be considered. Studies in children and adolescents show that the obese eat less than the nonobese but also are less active.[10,11] At the University of Edinburgh[12] studies of adults who were gaining weight showed that caloric intakes did not increase sufficiently to account for their weight gain and therefore a reduction of physical activity was postulated as the major factor. In general, we must assume that physical activity tends to diminish as excessive body weight increases.

The concept of reduced activity can be carried even further. It is known that the rate of metabolism during sleep is markedly affected by physical activity during the day and in active persons is roughly equal to the calculated basal value if the average of all sleeping hours is considered. In less active persons there is a more complete relaxation of muscle during sleep, and the metabolic rate falls approximately 10% lower. Dabney suggests a relationship between muscle tone and caloric need important in the person who is relatively inactive while awake.[13]

Another factor in the economic use of caloric fuel is insulation provided by adipose tissue. Quaade studied factors of insulation and oxygen consumption in relation to a standardized exposure to cold, using a hypothermic blanket thermostatically adjusted so as not to cause shivering. The metabolic increase in lean Danish subjects averaged 33%; in normals, 11%; and in the obese, 2.5%.[14] This may help to explain why some persons remain lean in spite of a copious caloric intake, whereas obesity may persist in the absence of gross overeating.

PATTERNS OF EATING AND LIPOGENESIS

Many calorie-conscious people tend to omit breakfast and lunch and concentrate on one meal in the evening. This does not insure reduction of calories, and this pattern of eating may make weight maintenance or loss more difficult. Many studies with experimental animals indicate that patterns of eating affect lipogenesis, probably through influence on specific enzymatic reactions. Cohn[15] observed that rats, force-fed to obtain their entire day's food in two meals, almost doubled body fat compared to animals nibbling the same quantitative diet at will. In addition, the force-fed animals showed decreased body protein and water, altered thyroid and tissue enzymatic activities, increased atherosclerosis, and increase in the severity of diabetes. Hollifield and Parson[16] showed that, when food was limited to one meal a day, rats became obese after ten weeks, the rate of lipogenesis increased 25 times over the normal, and activities of two enzymes involved in lipogenesis (G6PD and 6PGD) increased threefold. They postulate that this regimen would tend to perpetuate obesity in the human.

Once animals have become obese and then

have been reduced to normal weight, it is easier for them to become obese again. Using deoxyribonucleic acid content as an index of the number of cells in adipose tissue, there are a larger number of adipose tissue cells waiting to be refilled with fat.[17]

Evidence in human beings to support the concept of increased lipogenesis is beginning to appear. Since their extensive studies on rats had shown similar adaptive changes, Fabry and his colleagues in Prague surveyed 369 working males 60 to 64 years of age.[18] Those eating 3 meals a day or less were found to have more excess adipose tissue, higher plasma cholesterol levels, and lower glucose tolerance than the nibblers consuming 5 or more meals. In their view, a large meal in excess of immediate need stretches the metabolic mechanisms of disposal; repeated excess lipogenesis, even to a small extent, may damage these mechanisms. Four subjects in a metabolic ward at Ohio State University Hospital were fed an isocaloric diet for 3 separate two-week periods. The diet was provided as a single meal, 3 meals, and 10 feedings daily. Nibbling produced normal glucose tolerance test curves; gorging produced "diabetic" type curves. These alterations in configuration of the glucose tolerance curves appeared to be consistent with metabolic adaptations to increased gluconeogenesis and lipogenesis induced by infrequent feeding.[19]

Carbohydrate content of the foods consumed may influence both lipogenesis and fat mobilization. Studies employing C^{14}-labeled glucose indicate that obese subjects may not metabolize glucose normally, but dispose of it through increased lipogenesis.[20] Since the metabolism of glucose is closely linked with synthesis of fatty acids, triglycerides, and fats, an abundant supply of carbohydrate possibly exerts a powerful influence toward lipogenesis. In addition, it may suppress fat mobilization. Preliminary work suggests that the fat-mobilizing hormone of the pituitary is depressed by high carbohydrate intakes, appearing in the urine in those with a low carbohydrate intake.[21] When low-calorie diets with higher proportions of calories from protein and fat are given, initial weight loss appears more rapid than in diets with higher proportions of carbohydrate. This difference has usually been attributed to greater water loss. However, Kekwick and Pawan[22] have recently shown by carcass analysis in mice that this difference is due to loss of fat tissue, not total body water. These factors offer further explanations of the influence of carbohydrate upon body fat.

APPETITE AND SATIETY

The influence of carbohydrate on appetite is unclear. Is it a depressant or a stimulant, or are there individual differences in response between the obese or potentially obese and the lean?

Yudkin studied 18 obese subjects on a low-calorie diet with glucose given before meals; 12 others were given the same diet without the glucose. After six weeks, the average weight loss in the first group was 6½ lb., in the other group, 11½ lb. The glucose accounted for only 1 lb. of the weight difference between the two groups, and he concluded that the glucose stimulated appetite, the subjects supplementing their intakes from outside sources.[23] Since glucose or carbohydrate may not satisfy and may even stimulate appetite, Yudkin and Carey investigated carbohydrate restriction.[24] A small group of obese subjects was given a "high fat" diet restricted only in carbohydrate and all lost weight. Daily calorie intakes were reduced between 13 and 55% or between 200 and 1,900 calories; no subject consumed significantly more fat than before and 3 consumed less. If appetite were controlled by carbohydrate presumably these subjects would not have been satisfied on the diet, yet they were. When an obese person is satisfied by a 2,000-calorie regime consisting largely of fat and protein, and then if offered 1,000 additional calories in the form of carbohydrate, he not only accepts the carbohydrate addition but is likely to take more fat as well. Yudkin and Carey suggest that there is a considerable individual variation in the amount of carbohydrate one can eat without reaching satiety and postulate that obesity occurs in those whose appetites are not easily satisfied or may even be stimulated by carbohydrate. An extreme example of this is seen in carbohydrate addiction. Bloom and Clark[25] reported on four obese "carboholies" who responded impressively to carbohydrate restriction but, when carbohydrates were added to the diet, could no longer control their food intake and promptly gained weight. In contrast, we have found that high-protein supplementation gives rise to reduction in other food intake except in those with chronic weight loss.[26]

Yudkin also suggested that starches and sugars may behave differently, and a recent study by MacDonald supports this view.[27] In man, starch diets caused serum lipids to fall; sucrose produced a rise. Apparently, a relative excess of carbohydrate, at least with a low-protein intake, can give rise to derangement of lipid metabolism.

The complex metabolic problems of alcoholism are beyond the scope of this paper, but many

chronic alcoholics are obese. When alcohol is removed from the diet they tend to eat more than comparable nonalcoholics because they require significantly more total calories to maintain or gain weight.[28] The obese alcoholic preferentially metabolizes alcohol calories[29] and, by saving carbohydrate, develops a carbohydrate excess at a lower intake level than the nonalcoholic.

THE MECHANISMS OF APPETITE

In 1952 Jolliffe described an appetite center in the hypothalamus, the "appestat," which reacted in some way to a shortage of calories to promote hunger and cause a desire for food.[30] The postulated mechanisms for its actions fall into three general groups. Mayer advanced the glucostatic hypothesis, suggesting that it responds to the "glucose potential." When the arteriovenous difference in glucose level is high, hunger is suppressed; when it falls, hunger develops.[31] However, Penick and Smith reported that the maximal suppression of food intake was two hours after injection of glucose when the arteriovenous glucose differences were small.[32] Another suggested mechanism involves nervous stimuli from contractions of the empty stomach which stimulate the desire for food, the presence of food in the stomach being associated with satiety. Quaade and Juhl have produced satiety by noncaloric meals in fasted subjects.[33] A third factor involves thermal stimuli postulated to affect the "appestat" by heat developed by the specific dynamic action of food.[34] The present concepts have been well coordinated by Miller,[35] who suggests that appetite is subject to a multiple set of controls involving several parts of the brain, operating through sensory stimuli such as taste and stomach distension, as well as metabolic processes and psychologic factors.

Biochemical aberrations related to appetite and satiety may contribute to the development of obesity. Hollifield and colleagues studied 80 obese patients, three-fourths of whom had rarely or never experienced recognizable symptoms of hunger or satiety.[36] After a five-day fast in hospital, the amount of food required to produce a sense of satiety was quite small in relation to their prefast dietary intake. They consider that either the satiety centers in the hypothalamus became more sensitive to food eaten or a change in the peripheral tissue during fasting depressed appetite or increased satiety. In addition, activity of 2 enzymes of the hexose-monophosphate shunt pathway (acetyl coenzyme A and reduced-form triphosphopyridine nucleotide) were significantly reduced by fasting.

Study of factors in the adequacy, excess, or depletion of the body's stores of glycogen and fat must be further pursued. It appears certain that through habit the internal regulatory mechanism of appetite may be set at a higher level and become less responsive to the ingestion of food. Obesity is a condition which is not only self-perpetuating but literally self-engrossing. As we listen to patients it is tempting to suspect a thermodynamic paradox, but as we learn more about obesity, the validity of the first law is confirmed.

CONCLUSIONS

Known factors leading to the development and perpetuation of obesity may be summarized as:

1] A familial and possibly genetic predisposition, influenced by parental obesity

2] Maintenance of habitual eating patterns of young adulthood despite the reduction of basal needs with aging

3] Established eating habits with an excess of calorie intake over caloric need of social or psychological origin at all ages

4] Insufficient physical activity for calorie intake

5] Further reduction of physical activity as obesity develops

6] Decreased muscle tone in the inactive person which further lessens calorie need while awake or during sleep

7] Increased insulation by adipose tissue which reduces metabolic response to exposure to cold

8] Patterns of eating which in timing or amounts produce a metabolic adaptation to increased lipogenesis and alter the neuronal and humoral factors in appetite and satiety

9] Carbohydrate intakes in amounts which exert an influence toward lipogenesis and may stimulate appetite in certain individuals

10] Carbohydrate excess at proportionately lower intake in the chronic alcoholic

11] Propensity of the extra cells in adipose tissue to be refilled with fat after reduction of weight

12] Satiety centers becoming less sensitive to food consumed or the internal regulatory mechanism of appetite becoming set at a higher level.

Recommendations for the prevention of obesity are:

1] In those families where one or both parents are obese, special care from the prenatal period throughout life must be taken to reduce the nutritional assault

2] Overnutrition with accelerated growth and maturation should be avoided, although it is a socially accepted triumph today

3] Special care must be taken during infancy, first school years, and adolescence to prevent a pattern affecting future adult obesity from developing

4] The menarche, first pregnancy, and menopause are times of significant weight gain in the female, and dietary restriction becomes important

5] The "normal" weight gain of 10 lb. per decade after the age of 25 must be prevented

6] Increase of physical activity is a desired goal but, if impossible, must be associated with a fall in calorie intake

7] Since there is increasing evidence that carbohydrate, particularly in some obese individuals, does not lead to satiety but to increase of other food intake and that sugars may be more implicated than starches:

(a) carbohydrate reduction is specifically of value not only in the treatment of obesity but also in its prevention in the potentially obese and

(b) carbohydrate restriction is of particular significance in the obese alcoholic

8] Nibbling (5 meals per day) leads to a diminution of lipogenesis and weight gain and is a desired diet pattern not only in weight reduction but also in the prevention of obesity.

Finally, specific diet patterns at variance with many of those presently accepted are necessary in the treatment of obesity. More important, however, watchful care of the potentially obese with appropriate dietary and activity measures offers a positive approach for the prevention of this major cause of incapacitation and early death.

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Ascorbic Acid and Carbohydrate Metabolism. II

Effect of Supervised Sucrose Drinks upon Two-Hour Postprandial Blood Glucose in Terms of Vitamin C State

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An earlier report[1] that revealed a significant relationship between the cortisone glucose tolerance test and the plasma ascorbic acid level in the human being confirmed other published data[2–14] showing that increased blood glucose levels are inversely related to vitamin C state. This project is designed to pursue the subject and possibly cast light on cause-and-effect through a study of the effect of sucrose drinks upon blood glucose in terms of vitamin C state.

Method of Investigation

Thirty-nine presumably healthy junior dental students participated in this experiment. The age distribution is summarized in Table 1. It should be noted that the majority (90%) are in the third decade.

On a Monday, nonfasting (two hours post-breakfast) blood glucose[15,16] and plasma ascorbic acid level[17] were measured. The group of 39 subjects was then randomly divided. Twenty-three students received 100 gm. of chemically pure sucrose daily. Each participant reported at 7:45 A.M. and 1:30 P.M., at which time the student ingested 50 gm. of sucrose in solution. The remaining 16 subjects served as the control group. On Friday of the same week, nonfasting (two hours after breakfast and 50 gm. of sucrose) blood glucose and plasma ascorbic acid level were remeasured by the same technician, with no knowledge of the earlier findings or whether the subjects were in the sucrose-supplemented or nonsupplemented group.

TABLE 1
AGE DISTRIBUTION

Age in years	*Control group number and percentage*	*Therapy group number and percentage*	*Total number and percentage*
22	1 (6.3%)	0 (0.0%)	1 (2.6%)
23	4 (25.0%)	5 (21.7%)	9 (23.1%)
24	3 (18.8%)	5 (21.7%)	8 (20.5%)
25	2 (12.5%)	2 (8.7%)	4 (10.3%)
26	1 (6.3%)	4 (17.4%)	5 (12.8%)
27	1 (6.3%)	3 (13.0%)	4 (10.3%)
28	1 (6.3%)	1 (4.4%)	2 (5.1%)
29	0 (0.0%)	2 (8.7%)	2 (5.1%)
30	1 (6.3%)	0 (0.0%)	1 (2.6%)
32	1 (6.3%)	0 (0.0%)	1 (2.6%)
35	1 (6.3%)	0 (0.0%)	1 (2.6%)
39	0 (0.0%)	1 (4.4%)	1 (2.6%)
Total	16 (100.0%)*	23 (100.0%)	39 (100.0%)*
Mean	25.9	25.9	25.9
Variance	13.6	11.9	12.2
S.D.	3.7	3.4	3.5
Significance of the differences of the means		P>0.500	
Significance of the differences of the variances		P>0.250	

*Approximate

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TABLE 2

PLASMA ASCORBIC ACID DISTRIBUTION

Control group				Therapy group			
Case number	*Initial*	*Final*	*Difference*	*Case number*	*Initial*	*Final*	*Difference*
3286	0.59	0.48	—0.11	3285	0.90	0.86	—0.04
3287	1.20	1.12	—0.08	3288	0.95	0.92	—0.03
3290	0.06	0.31	+0.25	3289	0.06	0.25	+0.19
3296	0.49	0.49	0.00	3291	1.00	0.98	—0.02
3298	1.14	1.04	—0.10	3292	1.20	0.80	—0.40
3300	0.12	0.12	0.00	3293	0.59	0.80	+0.21
3301	0.76	0.76	0.00	3294	0.75	0.78	+0.03
3304	0.65	0.76	+0.11	3295	0.98	0.61	—0.37
3305	0.70	0.69	—0.01	3297	0.78	0.78	0.00
3306	0.58	0.48	—0.10	3299	0.76	0.81	+0.05
3307	0.81	0.76	—0.05	3302	0.65	0.58	—0.07
3308	0.48	0.65	+0.17	3303	0.48	0.48	0.00
3309	0.65	0.70	+0.05	3310	0.95	0.90	—0.05
3319	0.80	1.00	+0.20	3311	0.75	0.86	+0.11
3321	0.86	0.69	—0.17	3312	0.48	0.42	—0.06
3323	0.30	0.24	—0.06	3316	0.53	0.48	—0.05
				3317	0.53	0.42	—0.11
				3318	0.53	0.42	—0.11
				3320	0.90	0.80	—0.10
				3322	0.18	0.24	+0.06
				3324	0.30	0.18	—0.12
				3328	0.98	0.98	0.00
				3329	0.69	0.90	+0.21
Mean	0.64	0.64			0.69	0.66	
	P>0.500				P>0.200		
Percentage difference		0.0%				—4.4%	

RESULTS

Table 2 summarizes the distribution of plasma ascorbic acid levels for both of the groups at the initial and final visits and the differences between the visits. It will be noted that, overall, no statistically significant differences exist.

Table 3 is the summary of the changes in the blood glucose levels in the two groups. In the control group, the mean change was less than 1%. In contrast, a mean reduction of 13% was observed in the therapy group. More importantly, Table 3 shows that the blood glucose change was statistically significant ($P < 0.001$) only in the sucrose-supplemented group.

An analysis of the Figure shows that there is considerable variability in the blood glucose levels both in the control and in the experimental groups. For example, in the control subjects (open circles), 9 values at the second visit were less than at the first visit; 6 were higher at the final examination. In the experimental group, (dark circles) the Figure also shows 19 were lower and only 3 higher at the end of the experiment compared with the initial observations. It is, therefore, quite clear that considerable variability in blood glucose levels was observed.

In order to study the variability in blood glucose levels in terms of the vitamin C state, chi-square analyses were performed comparing near-equal groups of subjects with the lesser blood glucose variability between the two visits ($\leq$10 mg. percent) versus those with the greater change (>10 mg. percent) in the light of the relatively poorer vitamin C state (<0.70 mg. percent) versus better ascorbic acid levels ($\geq$ 0.70 mg. percent). Table 4 suggests that there is no difference in the control group. This is underscored by a $P>0.800$. In contrast, a like statistical analysis of the experimental group (Table 5) shows that the majority of patients with the relatively better vitamin C state ($\geq$0.70 mg. percent) showed the lesser blood glucose fluctuation following sucrose supplementation. The significance of these relationships is clearly shown by a $P<0.050$. The evidence presented here suggests that, with sucrose supplementation, the

TABLE 3

THE EFFECT OF SUPERVISED SUCROSE DRINKS UPON NONFASTING BLOOD GLUCOSE LEVELS

Control group				Therapy group			
Case number	*Initial*	*Final*	*Difference*	*Case number*	*Initial*	*Final*	*Difference*
3286	75	70	— 5	3285	76	72	— 4
3287	65	80	+15	3288	76	70	— 6
3290	77	63	—14	3289	65	77	+12
3296	76	57	—19	3291	92	85	— 7
3298	80	73	— 7	3292	77	63	—14
3300	92	90	— 2	3293	83	65	—18
3301	57	71	+14	3294	92	70	—22
3304	80	78	— 2	3295	85	60	—25
3305	91	60	—31	3297	92	65	—27
3306	83	80	— 3	3299	83	82	— 1
3307	80	82	+ 2	3302	92	70	—22
3308	75	84	+ 9	3303	77	77	0
3309	60	110	+50	3310	71	65	— 6
3319	73	83	+10	3311	92	85	— 7
3321	63	63	0	3312	75	63	—12
3323	95	80	—15	3316	117	80	—37
				3317	70	77	+ 7
				3318	84	60	—24
				3320	83	80	— 3
				3322	83	60	—23
				3324	127	103	—24
				3328	77	85	+ 8
				3329	85	83	— 2
Mean	76.38	76.50			84.96	73.78	
	P>0.500				P<0.001*		
Percentage difference	+0.16%				—13.16%		

*Statistically significant

TABLE 4

CHI-SQUARE ANALYSIS

(Control group)

	Nonfasting blood glucose level difference of >10 mg. percent	*Nonfasting blood glucose level difference of ≤10 mg. percent*	*Total*
Plasma ascorbic acid level ≥0.70 mg. percent	2	3	5
Plasma ascorbic acid level <0.70 mg. percent	5	6	11
Total	7	9	16

Chi-square = 0.0416

P>0.800

TABLE 5

CHI-SQUARE ANALYSIS

(Experimental group)

	Nonfasting blood glucose level difference of >10 mg. percent	*Nonfasting blood glucose level difference of ≤10 mg. percent*	*Total*
Plasma ascorbic acid level ≥0.70 mg. percent	3	8	11
Plasma ascorbic acid level <0.70 mg. percent	9	3	12
Total	12	11	23

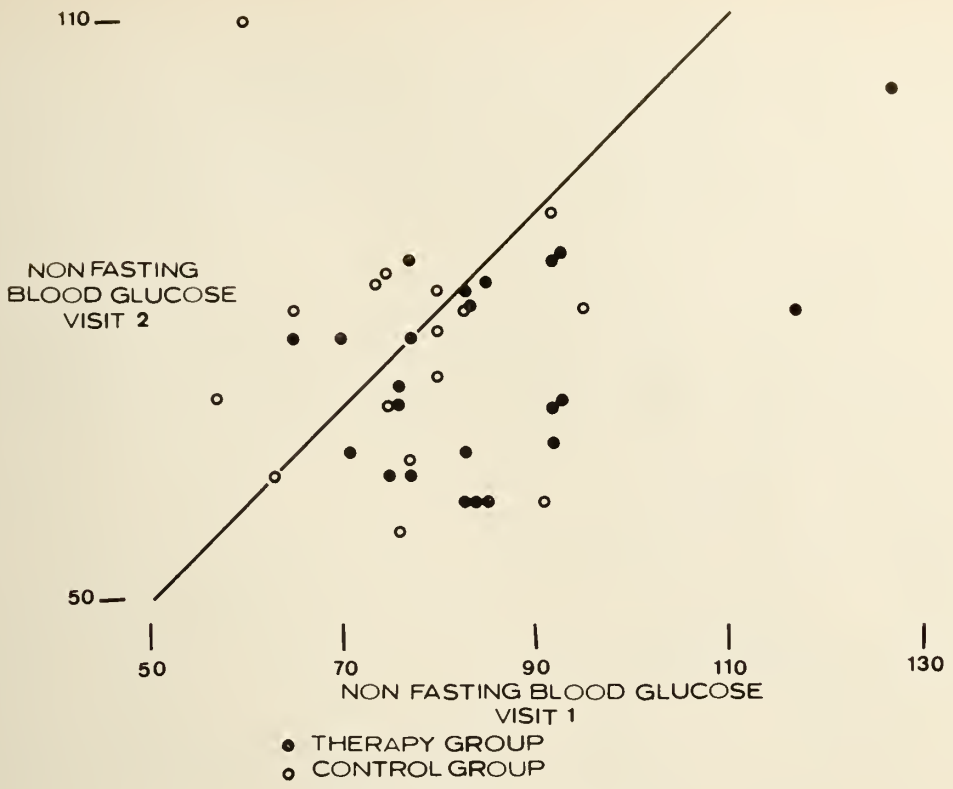
Chi-square = 5.2392

P<0.050

blood glucose level remains more steady in those individuals characterized by a relatively higher plasma ascorbic acid level. This is consistent with the earlier reported observation[1] of a significant relationship between carbohydrate metabolism (as measured by the cortisone glucose tolerance test) and vitamin C state (as measured by plasma ascorbic acid level).

SUMMARY

Thirty-nine presumably healthy junior dental students participated in an experiment to study



Variability in blood glucose levels

the effects of supervised sucrose drinks upon carbohydrate metabolism in terms of vitamin C state as reflected in plasma ascorbic acid concentration.

In the overall picture, the 16 subjects in the control group showed no change in nonfasting blood glucose; the group of 23 given supervised sucrose drinks showed a statistically significant reduction in nonfasting blood glucose. Chi-square analyses indicated that those with the least variability in blood glucose were characterized by higher plasma ascorbic acid levels.

A report to follow[18] will study the converse of the problem. Specifically, attention will be directed to blood glucose homeostasis following placebo versus vitamin C supplementation.

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Editor: JOHN S. LUNDY, M.D., Seattle, Wash.; *Coeditor:* RALPH M. TOVELL, M.D., West Hartford, Conn.; *Associate Editors:* ROBERT R. JONES, M.D., Rochester, Minn., JOSEPH A. GIBILISCO, D.D.S., Rochester, Minn., FLORENCE A. McQUILLEN, C.R.N.A., Chicago, Ill., LLOYD H. MOUSEL, M.D., Seattle, Wash., JOHN W. PENDER, M.D., Palo Alto, Calif.

Worthy of attention

LSD or d-lysergic acid diethylamide, a 1938 laboratory discovery dormant for five years, has become the subject of lively discussion in many quarters as to its affect on the mind of man. Phantom pain is also a considerable mystery. This discussion of such a phenomenon being treated by psychopharmacological means deserves attention. — JOHN S. LUNDY, M.D.

The Effect of LSD on the Phantom Limb Phenomenon

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THE CAPACITY of lysergic acid diethylamide (LSD) to induce delusions and hallucinations has been likened to the production of a "model psychosis."[1–3] It has also been established that following the intake of LSD the patient's body image changes before he is aware of any changes in his environment.[2] In view of these known effects, we determined to study the influence of LSD upon the sensory complex known as phantom limb "which is the most marked expression of the postural model of the body."[4]

In our study with LSD, we hoped to avoid the usual progression of symptoms by employing a small dose, thereby avoiding any confusional state or psychosis, while producing changes in body image. Our study involved 8 patients. Phantom limb occurred after brachial plexus paralysis in 1 patient, on the paralyzed side in 1 hemiplegic patient, and following limb amputation in 6 patients.

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CASE REPORTS

Case 1. On Feb. 10, 1961, a 26-year-old, right-handed, male factory worker fell from a height of 12 meters. He was comatose for ten minutes and was later found to have a motor paralysis and sensory disturbance of the right upper limb. When he regained consciousness after his accident, he was aware of a phantom limb and this symptom persisted. It was so intense an experience that he told his mother he had three hands. The phantom limb originated at the elbow joint of the paralyzed right arm and was directed inward and upward across the chest (Fig. 1). Its size approximated that of the actual right limb. Perception of the phantom limb was more vivid in the fingers than in the forearm.

The intensity of experience of the phantom limb varied with the position of the paralyzed right limb. When the latter was passively moved from the chest wall, the phantom limb was felt more distinctly. For this reason, the patient would passively move his paralyzed right limb from the chest wall with his left hand before discussing any symptoms of his phantom limb. He experienced paroxysmal pain in the phantom limb, the pain radiating upward from the finger tips. Voluntary movement of the phantom limb was almost impossible, but the fingers could be slightly flexed. There was no auto-

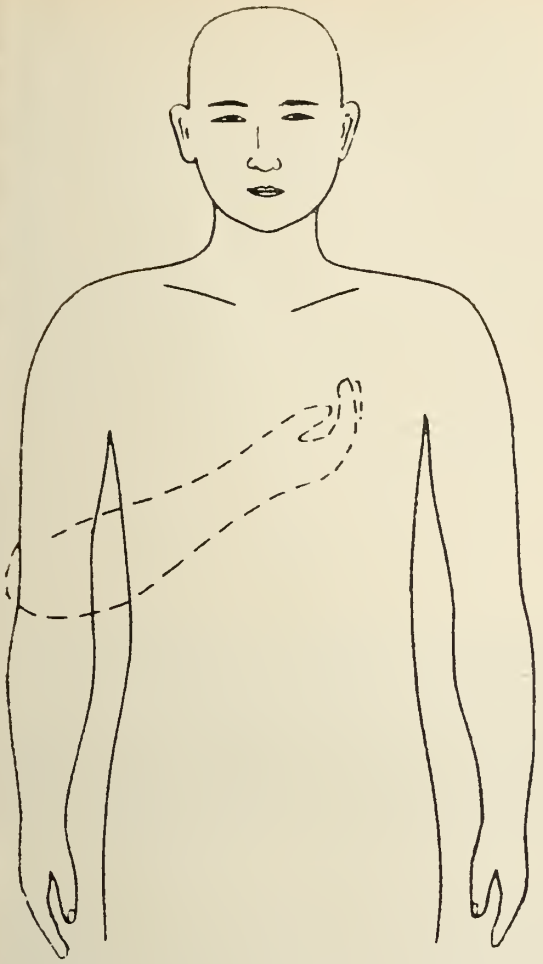


Fig. 1. Case 1: phantom limb shown by broken lines

matic movement of the phantom limb or synkinetic movement in the left hand when he tried to move the phantom limb. He did not express any feeling of negation regarding his paralyzed right limb.

Examination. The patient was alert, fully oriented, and cooperative. Abnormalities on neurological examination were confined to the right upper limb. Moderate muscular atrophy was observed in the right scapular region, right upper arm, and right forearm. There was complete paralysis of the right upper limb which was anesthetic. By passively increasing the distance between the chest wall and the paralyzed right upper limb, the phantom limb could be more clearly felt by the patient. An electromyogram supported a diagnosis of neural rupture between C4 and T1.

LSD experiment. On June 2, 1961, 50-μg. of LSD were administered to the patient. About thirty minutes later, the pain in the ulnar side of the phantom forearm was alleviated and the patient experienced a gradual shortening in the phantom forearm and palm. After one hour the phantom limb as a whole was felt more feebly. If the paralyzed limb was held close to the chest wall, the phantom limb was not experienced but returned weakly when the paralyzed limb was passively moved away from the chest. After one hour and a half the patient felt that the paralyzed and phantom limbs were superimposed, the phantom limb being felt more feebly than the paralyzed one. Occasional paroxysms of pain were experienced in the phantom limb. After two hours the forearm of the phantom limb was not felt and the patient was aware only of the palm and fingers. The paralyzed limb and right half of the body felt heavy. After two and one-half hours each finger of the phantom limb was felt separately, but finger shape and position were indistinct. After three hours the patient complained of intermittent, severe phantom limb pain which appeared superimposed on the paralyzed limb. After four hours the pain disappeared and the phantom limb was no longer felt, even when the paralyzed limb was held away from the chest wall. The paralyzed limb felt heavy. Gradually, the phantom limb sensation returned but was less vivid.

On June 16, 1961, another 50 μg. of LSD were administered to the patient. Since that time, passive movement of the paralyzed limb has not been accompanied by phantom limb sensation. The patient said of his phantom limb, "If I think it is present I feel it, but it is as if it were superimposed on my real arm; I cannot feel it distinctly." There was no recurrence of phantom limb pain. A sensation of weight persisted in the paralyzed right upper limb.

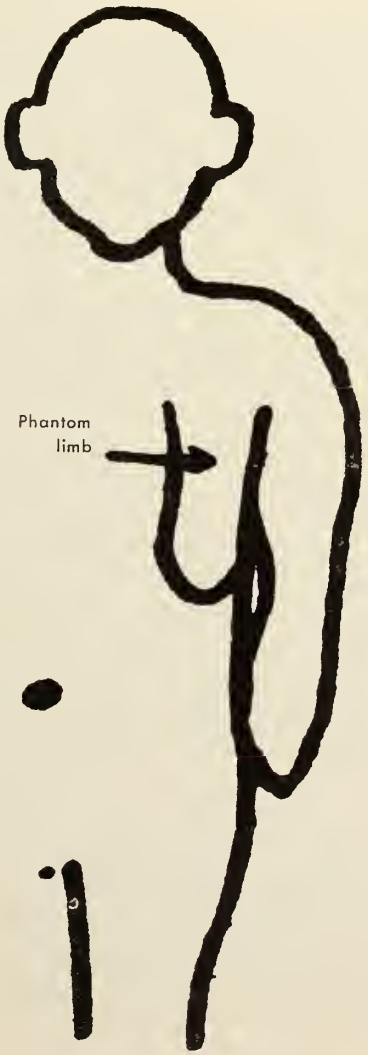


Fig. 2. Case 2: the patient's own sketch of phantom limb

Case 2. On April 15, 1957, a 58-year-old, right-handed male suddenly developed a left hemiplegia and was unconscious for three days. When he regained consciousness, he was aware of a phantom left arm and leg. The phantom arm felt particularly real and was, in fact, experienced more vividly than the paralyzed left upper limb. He was unable to determine the position of the latter unless he looked at it. Staring at the real limb did not dispel the phantom limb sensation. The phantom limb felt fixed and immobile, and the fingers and distal portion of the limb were recognized more distinctly than the proximal parts. He described the phantom upper limb as "like a clumsy hand of a child" and was able to draw a diagram of its position (Fig. 2). He complained of diplopia in January 1958 and was admitted to the hospital.

Examination. The patient was an alert, fully oriented male of average intelligence and normal personality. Examination of the cranial nerves revealed paralysis of conjugate lateral gaze to the right, right abducent paralysis, and a right facial palsy. There was a left hemiplegia and a left extensor plantar response. Sensation was intact apart from an inability to recognize the shape and texture of objects held in the left hand. Sensory stimuli (tactile, algesic, and thermal) applied to the left upper limb were perceived in the phantom limb (alloesthesia phenomenon).

LSD experiment. The patient was given 50 μg. of LSD orally. He remained alert throughout the experiment. Thirty minutes after taking LSD he felt that the paralyzed, left upper limb really existed and was capable of movement. After two hours and twenty minutes it felt light and he was able to actively move it across his body. However, he had the feeling that the phantom limb was moving the real limb. Tactile and painful stimuli applied to the real limb were now perceived and correctly located in that limb. After three hours and twenty minutes he felt that the real limb was independent of the phantom limb and was able to move itself. The phantom limb had become an encumbrance and, after four hours, seemed to be pressing against his chest, a reversal of the situation prior to LSD administration. After four and one-half hours the phantom limb sensation remained, feeling heavy, "like stones." It was felt most strongly in the wrist. These symptoms were present two weeks later when the patient was discharged from hospital.

The following six cases of phantom limb occurred after limb amputation.

Case 3. A 26-year-old, right-handed male injured his left leg in a traffic accident and amputation of the leg at the junction of the middle and lower thirds of the femur was necessary. Following amputation the patient was aware of a phantom limb and severe phantom limb pain which caused insomnia. On the tenth and fifteenth days following amputation, 50 μg. of LSD were given orally and the phantom limb pain was permanently

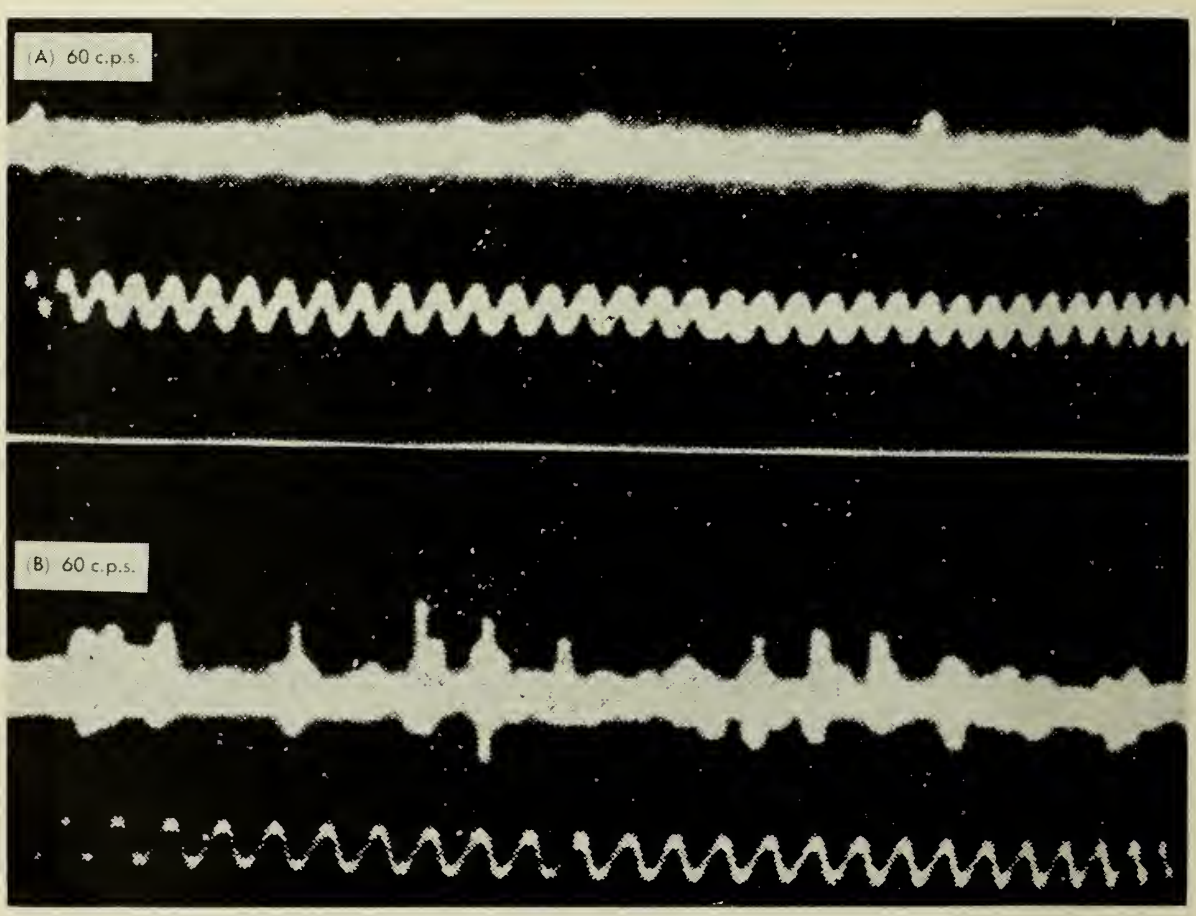


Fig. 3. Case 4: muscle action potential (muscle deltoids), [A] before LSD administration, [B] three hours after LSD administration

alleviated. The phantom limb sensation disappeared, subsequently returning with diminished intensity.

Case 4. A 23-year-old, right-handed male was injured in a traffic accident and amputation of the left upper limb 10 cm. below the elbow joint was necessary. Phantom limb sensation and severe phantom limb pain followed amputation. Eight months later, 50 μg. of LSD were given orally. After four hours the phantom limb sensation and pain disappeared completely, and the patient's only complaint was of difficulty in balancing his body since the loss of the limb. Subsequently, the pain returned but was not as severe and the phantom limb sensation was not as strong as before LSD therapy. During a period of sixteen months, 50 μg. of LSD were given three times. On each occasion the immediate beneficial effect did not persist, but the symptoms were eased. An electromyogram (EMG) was studied following the third LSD administration. The needle electrode was inserted into the left deltoid and the muscle action potential recorded. The results are shown in Figs. 3A and B. Muscle action was increased, indicating an apparent increase of motor activity.

Case 5. A 21-year-old, right-handed male injured his right lower limb in a traffic accident and amputation was performed 10 cm. above the knee joint. Six months later, 50 μg. of LSD were administered orally and a second similar dose after a short interval. Following this therapy the phantom limb sensation was almost completely eliminated and the phantom limb pain disappeared.

Case 6. Phantom limb sensation and weak phantom limb pain were experienced by a 26-year-old, right-handed male following amputation of the left lower limb below the groin. One year later, the oral administration of 50 μg. of LSD produced immediate and sustained disappearance of the phantom limb sensation and almost complete relief of phantom limb pain.

Case 7. A 26-year-old, right-handed male required amputation of the lower third of the right forearm following trauma sustained in a traffic accident. He was seen twenty months later complaining of vivid phantom limb sensation and phantom limb pain which had developed following amputation. It was also evident that the patient was a victim of some mental conflict. When 50 μg. of LSD were given orally, the mental conflict became clearly manifest. It appeared that he had experienced ambivalent, vehement feelings toward his

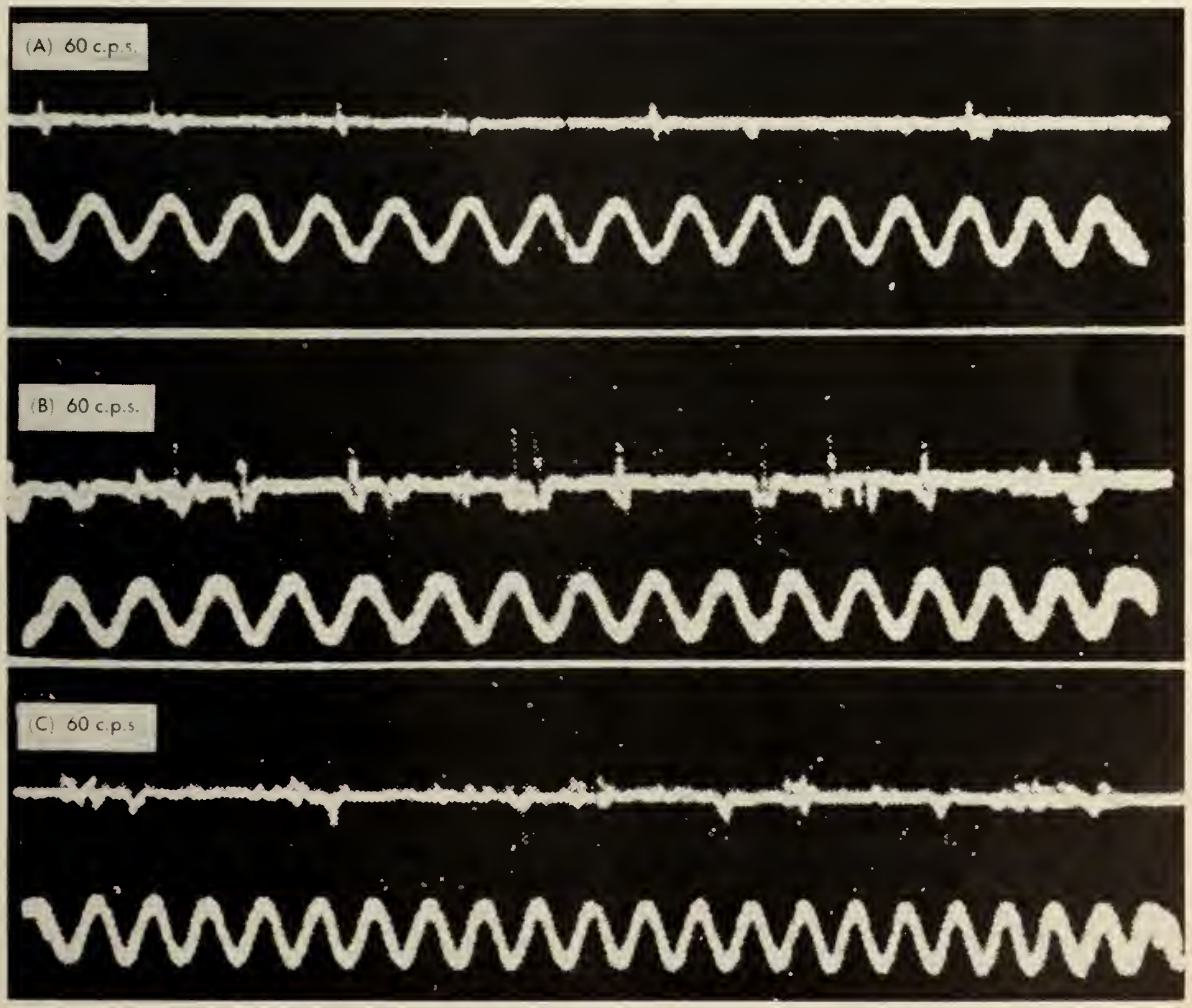


Fig. 4. Case 8: muscle action potential (medial rectus femoris), [A] before LSD administration, [B] three hours after LSD administration, [C] eight hours after LSD administration

dumb mother since his separation from his father at the age of 6 years. There was little change in the phantom limb sensation or the phantom limb pain. Following the treatment with LSD, his mental attitude improved and he was better able to bear the phantom limb pain.

Case 8. Phantom limb sensation developed in a 16-year-old male following amputation of the right lower limb 12 cm. above the knee for a right tibial sarcoma. Eight months after amputation an EMG of the left medial rectus femoris showed comparatively weak muscle activity and diminished discharges (Fig. 4A). Three hours after 50 μg. of LSD were given orally, the phantom limb sensation disappeared and the patient reported that the amputation stump had become light and easy to move. The EMG demonstrated stronger muscle activity and increased discharges (Fig. 4B). After eight hours, the phantom limb sensation returned faintly and the EMG showed decreased motor discharges (Fig. 4C), but these were greater than those obtained prior to the administration of LSD. The same results were obtained when LSD was administered on a subsequent occasion.

REVIEW OF CASES

It is noteworthy that the sensation of phantom limb in all 8 patients was not a transient symptom but a persistent complaint of a disturbing nature. It was felt more strongly in the distal parts of the limb. A small dose of LSD (50 μg.) produced significant effects in 7 (Cases 1, 2, 3, 4, 5, 6, and 8) of the 8 patients. A few hours after LSD was given, there was loss of the phantom limb sensation in 5 (Cases 1, 3, 4, 6, and 8) of these 7 patients, which was sustained in 1 patient (Case 6) and was much fainter on its return in the other 4 (Cases 1, 3, 4, and 8). In the remaining 2 patients (Cases 2 and 5) following LSD administration, the phantom limb sensation became much less distinct but persisted. LSD had no influence on the phantom limb sensation in 1 patient (Case 7) who was psychologically disturbed.

Phantom limb pain was a distressing symptom in 6 (Cases 1, 3, 4, 5, 6, and 7) of the 8 patients. Following LSD administration, it disappeared in 4 patients (Cases 1, 3, 4, and 5), permanently in 3 (Cases 1, 3, and 5), and was far less intense on its return later in 1 (Case 4). There was almost complete relief of pain in 1 patient (Case 6) and little change in Case 7, the psychologically disturbed patient. Apart from minimal changes in mood, no other effects on the psyche were noted with these small doses of LSD.

TIME SCHEDULE OF CHANGES IN PHANTOM LIMB PHENOMENA IN AMPUTEES FOLLOWING LSD ADMINISTRATION

Time after administration of LSD	*Experience of patients*
0.5 to 1 hour	Sensation of elongation of phantom limb. Recovery of normal sensation in proximal part of real limb
1 to 2 hours	Amputation stump felt lighter and was easier to move. Phantom pain replaced by itching
2 to 3 hours	Phantom limb sensation and phantom limb pain less distinct. Sensation of shortening of phantom limb
3 to 4 hours	Disappearance of phantom limb sensation and phantom limb pain

DISCUSSION

The phenomenon of phantom limb has frequently been observed after amputation of peripheral or external body parts, whereas it has not been reported following the removal of an internal organ. The same phenomenon has been seen in patients with a paralyzed limb. It would appear that the hallucination of a phantom limb counteracted sensations of body image disintegration.

Our study of the effects of LSD on the phantom limb experience revealed that there was a gradual transition from the moment when the phantom limb sensation altered to the time when it disappeared, as shown in the table.

Characteristically, phantom limb sensation after amputation was most strongly perceived in the distal parts of the phantom limb, whereas the sensation was weak or absent proximally in Cases 3, 4, 5, 6, and 8. This suggested that maintenance of the stable phantom limb sensation might be attributed to the fact that it was mainly represented by the functionally dominant part of the limb. One to two hours after LSD was given, the proximal parts of the limb, which hitherto had been felt faintly, were more distinctly perceived. Later, the phantom limb sensation diminished as a whole and, finally, in 3 cases, was no longer felt (Cases 4, 6, and 8). It may be theorized that LSD, by influencing the perception of body image, rendered the hitherto stable form of the phantom limb unstable and so capable of extinction.

Similar effects of LSD were observed in Cases 1 and 2. Phantom limb sensation in Case 1 accompanied a paralyzed arm, the result of brachial plexus paralysis. The intensity of the phantom limb experience varied with the position of the paralyzed limb, being greater when the latter was separated from the chest. One hour after LSD administration, the phantom limb was only felt if the paralyzed limb was held away from the chest. After one and one-half hours, the phantom limb and the paralyzed limb were felt to be superimposed, the phantom limb being more feebly perceived. The phantom limb was no longer felt after four hours, even when the paralyzed limb was held away from the chest. These facts suggested that the paralyzed limb was the background for the shape configur-

ation of the phantom limb. LSD, by influencing the patient's body image perception, caused changes in awareness of the paralyzed limb and thereby brought about the disappearance of the phantom limb.

In Case 2, the patient completely neglected the real paralyzed limb because of the vivid phantom limb sensation. Sensory stimuli applied to the paralyzed limb were felt in the phantom limb. This phenomenon of alloesthesia vanished following LSD administration, and stimuli applied to the paralyzed limb were accurately located in that limb. Simultaneously, voluntary movement of the paralyzed limb was possible and the phantom limb sensation gradually diminished. The effect of LSD was to incorporate the paralyzed limb in the patient's body image, thereby destroying the body image responsible for the phantom limb sensation.

EMG tracings at the amputation stump in Cases 4 and 8 following LSD administration, when the patient no longer felt the phantom limb, demonstrated an increase in electrical discharges compared with those recorded prior to the taking of LSD. The patient stated that the stump felt lighter and was easier to move. Once the phantom limb had been eliminated, all the patient's concentration could be devoted to the act of moving the stump which felt more real. After the administration of LSD in Case 2, voluntary movement was partially restored to the previously paralyzed upper limb which the patient truly felt existed while the phantom limb sensation was definitely weaker.

It has been reported that patients receiving LSD experienced decreased sensitivity to pain. Kast[5] found that LSD induced a more effective and prolonged analgesic effect than meperidine (Demerol®) or hydromorphone (Dilaudid®) in 50 terminally ill patients. He surmised that LSD altered the psychic elaboration of painful sensation. In our study, there was amelioration of phantom limb pain in 5 out of 6 patients following the administration of LSD.

LSD should be regarded as a useful drug in the treatment of phantom limb phenomena. It is hoped that these observations will provide a stimulus for further study.

CONCLUSION

In a dose of 50 μg., LSD benefited 7 out of 8 patients with phantom limb sensation and 5 out of 6 patients with phantom limb pain.

It has been postulated that LSD achieved these effects by changing the patient's body image and so destroying the psychological framework which was the genesis of the phantom limb sensation.

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Book Reviews on Pain . . .

HISTORICAL AND EXPERIMENTAL APPROACHES TO MODERN RESUSCITATION *by* JOHN W. PEARSON, B.M. *(Oxon.), 106 pp., Charles C Thomas, Springfield, Ill. 1965. $6.50*

This book is an effort, according to the author, of an attempt to change for the first time in two hundred years the title "First-Aid" to "Resuscitation" in the case of respiratory-circulatory failure. He archly states: "The famous American gynecologist, Sims, was demonstrating his operation for vesico-vaginal fistula in Paris in 1861 when his patient, under chloroform anesthesia, had a cardiac arrest. Sims did not know what to do, although the French surgeon Nélaton who was an interested spectator had definite ideas about resuscitation and was able to save the patient." The book is printed on good paper, it is indexed, and has a list of 121 references that have largely to do with cardiac or respiratory failure and resuscitation.

The early history of resuscitation is revealing to any reader. For example, Leroy-d'Etiolles' method of artificial respiration of moderate pressure on the thorax and upper abdomen with the patient in a supine position, was one of the forms of artificial respiration applied to Abraham Lincoln after he was shot. Modern methods are briefly described, and the experimental and research efforts that have contributed to modern methods are described and discussed.

This book should be of more than passing interest to people in anesthesia and those who are concerned with it.

JOHN S. LUNDY, M.D.

HYPERBARIC OXYGENATION *by* CHARLES B. PITTINGER, M.D., *113 pp., Charles C Thomas, Springfield, Ill., 1966. $5.50*

As the author says: "This monograph has been prepared to provide a concise introduction to the various aspects of hyperbaric oxygenation. The scope is broad in order to satisfy diversified interests and to provide to each a balanced view of the basic scientific and clinical implications of the subject.

Four categories of readers were considered: (1) those desiring merely a conversational acquaintance with the currently popular subject, (2) those with research inclinations voluntarily committed to its laboratory or clinical investigation, (3) clinicians, who perforce are confronted with its application or problems associated with it, and (4) students for whom the subject deserves to be a part of a progressive medical education.

"The information offered has been culled from widely scattered sources and compiled to provide a concise insight to the history, theory, scientific investigation, and clinical application of hyperbaric oxygenation, and appreciation of the attendant problems and complexities.

"The comprehensiveness of the bibliographies was intentional in an effort to provide within a single volume references to the scattered sources of pertinent information."

This book contains the following chapters: (1) Historical Development, (2) Physiologic Considerations, (3) Oxygen Toxicity, (4) Decompression Sickness, (5) Nitrogen Narcosis, (6) Clinical Applications: Status of the Literature, (7) Cancer Therapy, (8) Surgery, (9) Anaerobic and Other Infections, (10) Resuscitation, Poisonings, Asphyxia Neonatorum, Cardiac Arrest, (11) Cardiovascular Insufficiency, (12) Shock, (13) Hyaline Membrane Disease, (14) Hyperbaric Anesthesia, (15) Safety Considerations, and (16) Status of Hyperbaric Oxygenation.

The book is well indexed and the bibliography is remarkably usable for the active practitioner of today who must familiarize himself with hyperbaric oxygenation. It is fortunate that such a condensed version of this information is available.

JOHN S. LUNDY, M.D.

SCIENCE AND PRACTICE IN ANESTHESIA *edited by* JAMES E. ECKENHOFF, M.D., *160 pp., J. B. Lippincott, Philadelphia, 1965. $7*

In commemorating the Bicentennial Anniversary of the University of Pennsylvania School of Medicine in 1965, the Department of Anesthesia held a Reunion Refresher Course for its present and past residents, fellows, and staff. The papers presented at the meeting, some of which were rewritten, comprise the chapters of this book. All were prepared by alumni or current members of the department, with one exception—Dr. Christian Lambertsen. His long and close association with the anesthetists, as consultant, teacher, and collaborator, more than entitles him to membership in the department.

As the author stated in his Foreword, "Science and Practice in Anesthesia was not chosen casually as the title of this book. It was selected because it typifies historical activities related to anesthesia at the University of Pennsylvania School of Medicine. As far back as 1800, James Woodhouse, then Professor of Chemistry, demonstrated to the student physicians the effects in man of nitrous oxide. . . .

"Carl F. Schmidt, Professor of Pharmacology (1939-1959), was perhaps the first member of the modern faculty to see, in 1924, the future in fundamental investigation related to anesthetics; but the environment was inappropriate at the time, and he turned instead to respiration, in which he has long been eminent. At about the same time, I. S. Ravdin, Professor of Surgery (1945-1960), well aware of the inadequacies of the existing clinical administration of anesthesia, spent much of his time studying the relationship of anesthetics to liver damage. In 1938, both of these men saw in Robert D. Dripps the means of furthering investigation in anesthetics and of establishing an academic Department of Anesthesia. Alfred N. Richards, Professor of Pharmacology (1910-1939), then Vice-president for Medical Affairs, was the catalyst that finally led to Dr. Dripps' appointment in 1941 as Director of Anesthesia at the Hospital of the University of Pennsylvania.

Under Dr. Dripps' guidance, the department has

grown to be one of the outstanding such units in the world. Since 1945, over 265 physician anesthetists have participated in the department's instruction. The Reunion Refresher Course represents the teaching and activities of only a segment of that group. The subject matter was not meant to have continuity, but rather to represent areas of interest to practicing anesthetists, fields of endeavor of academic anesthetists and, in many instances, a posing of some of the challenges that face the specialty."

This indicates the character of the book. The subjects covered are interesting, and the material is up to date. The quality of the printing and paper is excellent, and it is indexed. It contains information of interest to all those in the field of Anesthesia.

JOHN S. LUNDY, M.D.

COMPREHENSIVE BACKGROUND FOR ANESTHESIOLOGY *by* W. FORREST POWELL, M.D., *346 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $15.75*

The author has attempted to present material which will help the student to learn and the teacher to instruct in the field of anesthesia. As the author says: "The material selected is designed to assist the resident in becoming an able clinician. Emphasis is put on the respiratory and circulatory systems, dysfunctions of which constitute most of the serious conditions that require informed supportive treatment during surgical procedures."

Subject matter of the topics is intended to serve as framework upon which the instructor may build complete formal conferences.

There is a minimum of space devoted to instruction in practical anesthesia administration.

"With few exceptions, little is contained in these pages that is entirely original.

"This volume is presented with the hope that the resident will be stimulated, by continual collateral reading, to become a competent consultant and perhaps a clinical investigator as well."

The presentation includes 121 illustrations, which were found necessary to give this outline more substance.

It is a book which should be in the hands of anyone who is preparing himself for examinations in the specialty and should be helpful to those who are preparing him for examination.

It is indexed, printed on good paper, and easily read. The field is given complete coverage.

JOHN S. LUNDY, M.D.

ANESTHESIA FOR OPEN HEART SURGERY *compiled and edited by* LILLIAN E. FREDERICKS, M.D. *and* DRYDEN P. MORSE, M.D., *80 pp., Charles C Thomas, Springfield, Ill., 1966. $5.50*

This book is made up as a symposium by 20 contributors and participants, mostly from Philadelphia, with 1 from New York and 2 from New Jersey. Two photographs of the group are included.

The overall important considerations are described very briefly, such as the importance of constant communication between surgeon and anesthesiologist and the importance of the problems that make this necessary.

The portion on intravenous procaine would have been improved by the proper use of the word "illness" instead of "sickness."

The outstanding chapter is on the choice of anesthesia on infants and children. The standard of excellence in this portion is not equaled elsewhere in the book, and this portion makes the whole book worthwhile. Nevertheless, the book generally is an excellent presentation for the benefit of those who have tried to watch the passing parade.

Fortunately, Raymond S. Barnes has contributed the information usually missing in such a presentation as this, and explains the problem of obtaining the blood needed for open heart surgery.

Large, easily read print has been used for this indexed book.

JOHN S. LUNDY, M.D.

DEATH IN THE OPERATING ROOM *by* ANTONIO BOBA, M.D., *105 pp., Charles C Thomas, Springfield, Ill., 1965. $5.50*

This unusual, small book is provocative in setting forth the difficulties of being at all certain of the cause of death in the operating room. It causes the reader to think—which is quite an accomplishment in itself. Because of its brevity and the inclusion of cases of significance, it should be of interest not only to people in anesthesia, but also to the other members of the operating room team and all who are concerned with the statistical significance of the findings.

JOHN S. LUNDY, M.D.

THE MAYO LEGACY *by* GUNTHER W. NAGEL, M.D., *157 pp. ill., Charles C Thomas, Springfield, Ill., 1966. $6*

The author writes truthfully of the period he spent as a fellow in the Mayo Foundation and the Mayo Clinic. His story is interestingly written. His conclusions concerning the institution and the personnel there are charmingly and honestly told. Fortunately, he has included remarks about the early history of surgery and nursing that are revealing to those who did not have the author's intimate view of the Mayo Clinic.

This book is recommended to those who would like to know the true nature of the Mayo legacy.

JOHN S. LUNDY, M.D.

FUNDAMENTAL CONSIDERATIONS IN ANESTHESIA *by* CHARLES L. BURSTEIN, M.D., *3rd ed., 223 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $8.50*

The foreword is by the late E. A. Rovenstine, M.D., under whom Dr. Burstein had his training, so that one can be sure that the included considerations in anesthesia are really fundamental.

In this third edition "two new chapters have been added to describe problems relative to adrenocortical insufficiency and complications of other newer drugs." The organization of the contents is on the basis of anatomical systems, emphasis being placed upon those considerations which have impressed the author. The text bears the field mark of competency and experience.

JOHN S. LUNDY, M.D.

Gaylord W. Anderson, M.D.

STEWART C. THOMSON, M.D.
Minneapolis



DR. GAYLORD WEST ANDERSON, director of the School of Public Health of the University of Minnesota and Mayo professor, has literally been a part of the university community since the day of his birth, December 31, 1901. He was born in a house which stood at the corner of University Avenue and Seventeenth Avenue S.E. in Minneapolis. The Beta Theta Pi fraternity house is now located on that site.

His parents, Prof. Frank Maloy Anderson and Mary Gertrude Steele Anderson, had lived in Minneapolis since childhood; his father came here in 1875 and his mother, in 1877. Both were 1894 graduates of the university, and it has been said that their wedding ceremony in 1898 was the first held in the present Westminster Presbyterian Church on Nicollet Avenue, then a new edifice. Professor Anderson was a member of the Department of History from 1894 to 1914, and former students remember him as a prominent faculty member and an active participant in Minneapolis civic affairs.

Dr. Anderson has often recalled that, as a small boy, he knew William Watts Folwell, the university's first president. Since then, he has known almost all the presidents of the university. Because his parents were active in the university community, Dr. Anderson has known faculty members who to most of us are only names in James Gray's history of the university written during the centennial in 1951. The old drill field on University Avenue, where the Museum of Natural History and the Center for Continuation Studies were later erected, was a favorite playground for Dr. Anderson and his brother, the late Prof. Troyer S. Anderson. Thus, Dr. Anderson's associations with the university really extend over almost sixty-five years.

He also has connection with the early medical history of Minneapolis. His maternal grandfather, Dr. John A. Steele, was a Minneapolis physician from 1877 until his death in 1911 and was active in community affairs.

In 1914, Professor Anderson received an appointment as professor of history at Dartmouth College, a position he held with distinction until his retirement in 1941. Thus, when Dr. Anderson was in his thirteenth year his family moved to Hanover, N.H. He graduated from Worcester Academy in 1918 and still can repeat parts of the Latin oration he gave at the commencement day exercises. He then attended Dartmouth College, from which he received the A.B. degree summa cum laude in 1922, with honors in chemistry and German. The following year, he studied at the Sorbonne, in Paris, and the University of Zurich. From 1923 to 1924 he was a teaching fellow in chemistry at Harvard University. He began his medical studies there in the fall of 1924 and received his M.D. degree cum laude from Harvard in 1928. Some years later, in 1942, he was to receive his doctorate in public health from Harvard.

Dr. Anderson interned at the Albany Hospital, Albany, N.Y. In 1929, he was appointed epidemiologist in the Division of Communicable

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Diseases, Massachusetts Department of Public Health; subsequently he became assistant director and, later, director of that division. From 1931 to 1937 he was deputy commissioner of public health in Massachusetts and during the same period was an assistant in public health administration at the Harvard School of Public Health. From 1935 to 1937, he served as executive secretary of the Massachusetts Legislative Commission to Investigate Public Health Laws and Policies.

In 1937, Dr. Anderson was invited to become professor and head of the Department of Preventive Medicine and Public Health at the University of Minnesota Medical School. Thus, after twenty-three years mainly in New England, he returned to the community in which he lived as a boy. With the reorganization of the Department of Preventive Medicine and Public Health into a School of Public Health in 1944, he became the founding director of the School of Public Health. In 1946, he also received the distinction of being the first person named to the newly created Mayo professorship. The development of the new School of Public Health was under Dr. Anderson's capable guidance, and it became a school of international reputation in which Dr. Anderson's vision of the true mission of such a school was realized.

From 1942 to 1945 he was on leave of absence from the university while he served with the Army Medical Corps in the surgeon general's office in Washington, D.C. He first was in charge of venereal disease education and later became director of the Division of Medical Intelligence. He advanced to the rank of colonel and was decorated with the Legion of Merit.

With Miss Margaret G. Arnstein, then director of public health nursing at the University of Minnesota, he coauthored a widely used textbook, *Communicable Disease Control*, published in 1941. In writing the fourth edition of the book in 1962, Mrs. Mary R. Lester of the Communicable Disease Center, Atlanta, Ga., assisted. The current edition is available in both French and Spanish. Dr. Anderson collaborated with Drs. James S. Simmons, Tom F. Whayne, and Harold M. Horack in writing *Global Epidemiology*. The series of three volumes, written in 1944, 1951, and 1954, covers the geography of disease and sanitation.

Dr. Anderson wrote the chapter on "Regulation in Public Health" in Graham and Reining's *Regulatory Administration* (1943) and the chapter on "The Principles of Epidemiology as Applied to Infectious Diseases" in the recently published fourth edition of *Bacterial and Mycotic Infections of Man*, edited by Dubos and Hirsch. He is the author of numerous articles in professional journals in the fields of public health administration and epidemiology. His special areas of interest and study in epidemiology have been the streptococcal diseases, typhoid fever, and poliomyelitis.

He has long been a member of the Committee on Communicable Disease Control of the American Public Health Association. In fact, he is now the senior member in years of service on this committee which every five years prepares the manual on *Control of Communicable Diseases in Man*. It is now in its tenth edition. Dr. Anderson has been associated with the preparation of the last five editions of this manual which is known and used throughout the world and which has more printed copies than any other work on public health.

An active member of the American Public Health Association since 1930, Dr. Anderson was chairman of the epidemiology section and elective councilor for three separate terms. He was a member of the Committee on Professional Education for eight years, a member of the Technical Development Board for a period of two years, and chairman of the Lasker Awards Committee. He also has served in other capacities. He was president of the American Public Health Association from 1951 to 1952. During this time, he was president of the American Epidemiological Society and the Association of the Schools of Public Health. On November 12, 1963, he was signally honored at the annual meeting of the American Public Health Association in Kansas City, Mo., by being the recipient of the Sedgwick Memorial Award for 1963, the association's highest award.

Dr. Anderson is well known as a consultant to universities on problems related to professional education of public health personnel as well as a consultant to various agencies on epidemiological problems. He has served as a special adviser to the World Health Organization (WHO) on public health manpower. He was a member of the United States delegations to the World Health Assembly in 1951 and 1958. He served as a special consultant to the Department of State in missions to Brazil, Argentina, Chile, and Peru in 1948 and to Colombia and Ecuador in 1949. These missions took him to Chile again in 1950 and to Korea in 1954. In the interests of medical and public health education, he has represented WHO in Egypt in 1953 and India, Iran, and Egypt in 1958. In 1963 he visited England, Scotland, Sweden, the Netherlands, and Yugoslavia with a group of public health leaders.

In the winter of 1965, he was on a WHO mission to Egypt, Lebanon, and Turkey.

His international interests are not his sole pursuits. Dr. Anderson also has served national, state, and local agencies. At present, he is chairman of the Task Force on Environmental Health of the National Commission on Community Health Services. His many contributions to this community in the field of public health were recognized in 1959, when the Minneapolis Junior Chamber of Commerce conferred upon him the Harrington Award. No problem has ever been too small for consideration when his advice has been sought on the matter. Other cities and states have frequently requested his help on special health problems. Through television and radio appearances as well as meetings of community organizations, many of the general public have come to know him. He is well known in the field of public health locally, nationally, and internationally.

Not only is Dr. Anderson an able administrator and a consultant on public health, but he is an outstanding teacher. Chaucer, in the general prologue to the *Canterbury Tales*, wrote of the Oxford clerk and said of him that "gladly wolde he lerne and gladly teche." These words, written almost six hundred years ago, are equally applicable to the truly great teachers of today. Among them is Dr. Anderson, who thoroughly enjoys his teaching duties. His students in all sections of this country and in many other countries remember him as a teacher of marked ability and as one who always found time, even on busy days, to talk over questions which came up in their studies. His presentations of what to many might be considered dull topics are enlivened by a rich humor—the kind of humor which William J. Long once described as "wisdom smiling"—and by appropriate examples drawn from his unique background in public health fields. Students soon learn that he holds and demands a high standard of scholarship. For this, they and his colleagues on the faculty have deep respect.

During his years at the University of Minnesota, Dr. Anderson has participated in the work of university-wide committees as well as those in the College of Medical Sciences. He has served with great diligence as chairman of the Senate Library Committee and the Bio-Medical Library Committee. It is remembered that his father, during his years on Minnesota's faculty, had had a special interest in the library. A member of Phi Beta Kappa, Sigma Xi, and Alpha Omega Alpha, Dr. Anderson has served as president of each of these honorary organizations. He also has been the president of the Campus Club.

Dr. Anderson is a diplomate of the American Board of Preventive Medicine and was one of its original trustees. He is an honorary fellow of the Royal Society of Health of England and has memberships in the American College of Preventive Medicine, American Epidemiological Society, American and Minnesota public health associations, Massachusetts Medical Society, American Medical Association, Phi Beta Kappa, Sigma Xi, Alpha Omega Alpha, Delta Omega, and Alpha Chi Sigma.

Dr. Anderson's marriage to Miss Viola Dennis of Newton, Mass., occurred on October 26, 1929. Their daughter, Gail, now Mrs. Harvey Safeer, lives in Springfield, Va. Two granddaughters, Jennifer Gail and Robin Louise, come from the East to spend holidays with their grandparents—visits which are eagerly anticipated by both the grandparents and the young ladies.

Students and faculty associates alike have enjoyed the gracious hospitality of Dr. and Mrs. Anderson. The annual reception which the Andersons hold at their home for students and faculty after the June commencement exercises has become a delightful tradition in the School of Public Health. Foreign students have gratefully remembered the pleasant occasions when they were guests at 2261 Folwell in University Grove. Some years, they have received visitors from two dozen countries. Every Christmas, the Andersons send greetings to each of the several hundred foreign students who have been enrolled in the School of Public Health, and in return, letters and cards come to Dr. and Mrs. Anderson from former students in almost all parts of the world. They have not forgotten the kindnesses extended to them during their months of study at Minnesota.

Dr. Edward S. Rogers, professor of public health and medical administration, University of California at Berkeley, presented the Sedgwick Memorial Medal for 1963 to Dr. Anderson. One of the paragraphs from the citation can well be used as a summary paragraph for this sketch. Dr. Rogers said:

> Over the years you have earned the deep respect and affection of all who have been fortunate enough to come under your guidance as an inspiring teacher; to encounter your keen insights and meticulous standards as an administrator; to feel the powerful influence of your wisdom as a statesman; or just to know you as a friend.

His former students, his faculty colleagues, and all others who have been privileged to know Dr. Anderson wholeheartedly endorse Dr. Roger's words.

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DORSEY Season

A journal within a journal published quarterly in the interests of better medicine by Dorsey Laboratories, a division of The Wander Company, Lincoln, Nebraska. Address communications to Raymond C. Pogge, M.D., Director of Medicine.



this issue: the nose as a shock organ

The nose as a shock organ

by Charles J. Shagoury, M.D., Chelmsford, Massachusetts



"Is it a cold, hay fever, or has he been reprimanded by his boss?" Occasionally, you will ask yourself this question when confronted by a patient with abrupt onset of rhinorrhea, nasal obstruction, and sneezing. Usually the history will elucidate the problem, but examination of the nose will often provide valuable clues to the correct diagnosis.

The nose is a shock organ in a double sense. First, it is in the nose that the confrontation takes place with the surrounding atmosphere. For twenty-four hours a day, the nose must meet the varying challenges of the inspired air, containing perhaps noxious chemicals, dust, dirt, bacteria, viruses, fungi, and industrial pollutants of all kinds, and render it clean, virtually sterile, and fit for the sensitive alveoli of the lungs. Whatever the temperature or humidity of the atmosphere, the nose must transmit it to the lungs at approximately 98°F, and with a humidity of approximately 40%.[1]

Second, in particularly susceptible patients, the nose acts as a shock organ in a manner totally unrelated to its normal function. Persons with hay fever respond to ordinarily harmless materials by extreme nasal congestion, with marked rhinorrhea and violent spasms of sneezing. In some patients, exposure to threatening or disagreeable agents, or situations involving mental conflict may result in a reaction which is exclusively nasal, with swelling of the turbinates, and marked hypersecretion.[2]

Nasal symptoms usually result when the nose seeks to perform its function of getting rid of noxious and dangerous elements in the atmosphere, and prevent their admission to the trachea and lungs. Small particles are removed by the mucous coating which blankets the nasal passages. This mucous blanket contains a bacteriostatic agent, lysozyme, which destroys most air-borne bacteria.[3] The mucinous content renders the surface sticky, causing dusts and small particles to adhere. It has been postulated that this process is rendered more effective through adsorption because of a surface electrical charge on the nasal mucosa.[4] The cilia then sweep the particulate matter to the pharynx. The nose can prevent entrance into the lungs of particles as small as three microns in diameter, but smaller particles elude the nasal barrier. Most bacteria causing respiratory infections are one to three microns in diameter, but since they usually are inhaled in clumps, they are efficiently removed as a rule. Viruses, which are of the order of 1/1000 of this size, are less efficiently dealt with, unless they occur in very large aggregates.[5]

The nose will react in a more or less similar manner, whatever the nature of the offending agent, whether it be an irritant chemical, virus, pollen, or distasteful emotional situation. In acute coryza, the most characteristic sign is a profuse watery discharge. The volume of secretion may rise from practically nothing to nearly 60cc in twenty-four hours.[6] The mucous membrane is reddened and engorged, while the turbinates are markedly swollen. After the first day or two, the secretion becomes thicker, yellowish, and more difficult to expel. The surface cells are largely destroyed, contributing to the copious discharge, which now also contains numerous inflammatory cells which have migrated to the area. Gradually, over a period of a few days, or a week, the flood abates, the swelling and redness subside, and the nasal epithelium resumes a healthy appearance.

Repeated attacks of rhinitis, particularly if there is an underlying element of obstruction, may result in chronic rhinitis. The mucous membrane is constantly swollen and reddened. Sticky, mucopurulent secretions are a continuous feature, and the glandular elements are hypertrophied. Commonly, the mucosal surface takes on an irregular, rounded "mulberry" appearance, and nasal passages are occluded by the swollen turbinates and redundant mucosa.

While all of us are susceptible to colds, the victim of hay fever, or allergic rhinitis, displays a marked nasal reaction to materials in the air which leave his associates unaffected. In such a patient, the nasal mucosa has become an allergic "shock" organ. Contact with the nasal allergen causes local release of histamine, with vasodilatation, increased vascular permeability, and severe nasal congestion, similar to the "wheal" and "flare" reactions in the skin, when the epidermis is the allergic shock organ. While we eagerly await the coming of spring, the hay fever sufferer dreads the blooming season, whose invisible pollens are poisons to his sensitive nose. His neighbor's cat or dog may provoke paroxysms of uncontrollable sneezing. In some cases a specific allergen is not identified, but the triad of rhinorrhea, nasal obstruction, and sneezing is present.[7] The nose in these cases shows a pale, boggy, edematous mucosa, with a thin mucoid secretion. The mucous membrane shows extreme retractility to 1% cocaine or ephedrine. If the patient has medicated himself prior to examination, the nasal passages may appear abnormally patent, or show exaggerated congestion due to rebound reaction. The secretion may show a large number of eosinophils particularly after an attack of sneezing or rhinorrhea. Touching the mucosal surface, especially of the inferior turbinate, leaves an indentation, showing that the swelling is due to stasis and edema, rather than actual hyperplasia of the mucous membrane as in chronic hypertrophic rhinitis. Though the pale swollen mucosa is the hallmark of allergic rhinitis, as usually seen by the physician, exposure of allergic subjects to their known allergens results in a brief hyperemic phase, followed by pallor and edema.[8]

In the later stages of allergic rhinitis, the chronic edema of the mucous membrane results in the formation of polyps, clusters of grape-like masses hanging from the roof of the nose, with a pale glistening surface, contributing significantly to the sense of nasal obstruction and oppression.

A large group of patients show symptoms of nasal congestion when confronted by adverse life situations.[9] In these unfortunate persons, anxiety, frustration, and resentment are often accompanied by a runny nose and nasal obstruction. Lacrimation adds to the nasal stuffiness. This autonomic response, mediated by the parasympathetic nervous system, may be part of a general parasympathetic reaction, or may possibly represent in part, a symbolic effort to wash out and crowd out the offending situation.

Nasal congestion may also occur in some patients at times of sexual stimulation, and in women during menstruation and pregnancy, even to the point of epistaxis.[10] The relationship is obscure; castration results in atrophy of the nasal glands, and their action is inhibited by the hormones of the hypophysis and the thyroid.[11] The nose may be the shock organ in drug therapy. The nose may also bear the brunt of industrial stress, in those who work in a hot dry atmosphere, or those exposed to acid fumes, or irritating dusts. As the air in our cities is increasingly polluted by exhaust fumes, and industrial irritants, whole urban populations may suffer from chronic nasal and respiratory symptoms.

Of course, nasal reactions are not just infectious, or allergic, or emotional. Particularly in the chronic sufferers, there is an interdependence of all three. Death of a relative, or other psychic shock can pre-

(concluded on following page)

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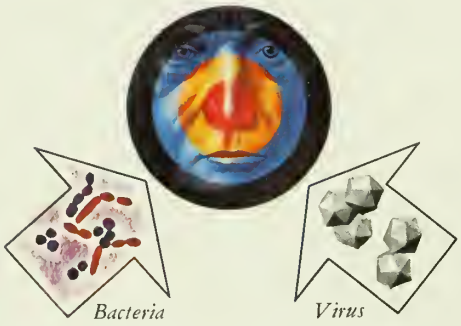
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cipitate an attack of rhinorrhea in hay fever sufferers.[12] Others develop attacks of vasomotor rhinitis following change in temperature, chilling, or exposure to the sun, or simply warm bedclothes.

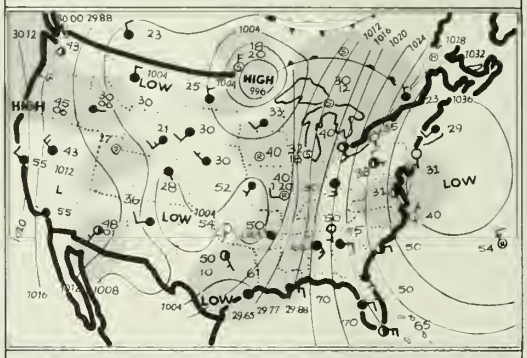


The complex interplay of allergy and infection is largely unclear. Allergy to the viruses and bacteria which cause infection has been postulated, but is difficult to demonstrate. The swollen obstructed allergic nose is more susceptible to infection. At the same time, infection often precedes or precipitates an allergic attack. Exposure of a susceptible patient to an allergen can activate latent virus organisms leading to infection.[13] This "jolt" reaction represents a summation of an allergen and a virus leading to symptoms in the nose as a shock organ, which neither could have produced alone. In childhood, repeated attacks of bronchitis and colds may be inflammatory reactions to an allergen, or precipitated by exposure to an allergen. These children may later develop typical allergic rhinitis. On the other hand, children with typical allergic histories, eczema, asthma, and allergic familial backgrounds, may later develop typical infectious rhinopathies. Skin tests in such patients are usually positive.

Nasal reactions are part of the systemic response of the patient to an unwelcome stimulus. In cases of respiratory infection and exposure to atmospheric irritants, the reactions are useful, and to some extent desirable. They are usually self-limited, disappearing within a few days, or upon removal of the provoking agent. Here the distressing symptoms can be ameliorated with appropriate decongestant agents, or, in the case of severe or complicated respiratory infections,antibiotics may be given, with reasonable confidence of a cure. On the other hand, when nasal reactions are the peculiar response of an individual to an allergen, or to an undesirable situation, they serve no useful purpose. The nose here is a shock organ in a stressful situation, but can furnish no response of value. It merely causes the patient symptoms which add to his problems. In these cases, symptomatic treatment is of great benefit, but often the underlying faulty pattern of response cannot be altered. Such a patient may literally be considered to be paying his way in life "through the nose."

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Book Reviews . . .

Endocrine Surgery in Human Cancers

by PAUL JURET, M.D., *2nd ed., (translated from original French by Ranes Chakravorty and Ernest Greenbert) 412 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $15.50*

This is the second edition of a book relating to endocrine surgery in human cancers. The author might better name the publication, since 278 pages are devoted to the endocrine aspects of breast cancer, 55 pages to prostate cancer, and only 5 pages to an inadequate coverage of other endocrine surgical factors.

The author carries out a review of the endocrine aspects of breast cancer and introduces his own concept of the role of this therapy in the treatment of breast cancer. This includes castration, adrenalectomy, hypophysectomy, and administration of estrogenic, androgenic, and progestational hormones. The larger and more significant studies have been analyzed and carefully considered by the author in the process of defining his concept of the role of these therapies in an established protocol system of treatment. A short section on the management of male breast cancer is disappointing.

A section on endocrine surgery in prostatic cancer is well reviewed, though little new material is available in this aspect of endocrine cancer therapy. This publication would be of most interest to those scientists interested in the hormonal therapy of breast cancer.

B. J. KENNEDY, M.D.
Minneapolis

International Series of Monographs in Pure and Applied Biology/Zoology Division: Neurosecretion

by M. GABE *(translated by R. Crawford), Vol. 28, 872 pp., ill., Pergamon Press, New York, 1966. $30*

It is hard to realize that neurosecretion, a problem studied extensively today, was looked upon as a morphological curiosity only a generation ago. The extent of interest now is shown by the vast number of papers on this subject published in the past fifteen years. Dr. Gabe has done a magnificent job in bringing together a summation of the work on the neurosecretory organs in the whole animal kingdom, giving equal weight to morphology, histology, and cytology on the one hand and physiology on the other.

The book is divided into 3 parts. Part I, General Considerations, is a methodical discussion of the concept of neurosecretion and the methods used in its study. Part II, Analytical Study, the "meat" of the book, covers in 43 chapters the work that has been done. Its division into 2 sections—Invertebrata and Vertebrata—may not be necessary with regard to zoological classification but by custom represents the normal. Each chapter is brief but gives sufficient detail. The section on Vertebrata covers the hypothalamo-neurohypophyseal neurosecretory pathway in 2 divisions—morphology and physiology. A third division discusses other neurosecretory phenomena. Part III, The Composite Picture, presents a "synthesis in which the aim has been to identify the general characteristics of neurosecretory cells and to determine the place of neurosecretion among the main animal functions." The final chapter in this part, Outlook and Conclusion, is an admirable statement of what has been and probably will be.

The book is written in clear, concise language (a tribute to the translater) and is completely readable. The illustrations are clear and well reproduced. The bibliography is comprehensive and extensive, containing more than 2,200 references. Biologists, anatomists, zoologists, and medical men owe Dr. Gabe a vote of thanks for this tremendous undertaking.

It is a highly specialized book. Those who merely want to know in a general way what neurosecretion is will not need it, but the researcher in this field cannot afford to be without it. This will be his "bible" for years to come.

DAVID B. SABINE
Yonkers, N.Y.

Resuscitation of the Newborn Infant and Related Emergency Procedures: Principles and Practice

edited by HAROLD ABRAMSON, M.D., *2nd ed., 411 pp., ill., C. V. Mosby Co., St. Louis, 1966. $16.50*

The title of this book is somewhat misleading, as most practitioners consider resuscitation to mean the restoration of life from apparent death by rapid establishment of the respiratory mechanisms. The authors, however, feel, and in my opinion rightly so, that this represents only the initial step in the restoration of breathing and that to maintain the initial advantage early diagnosis of the causes underlying delay in the onset of respiration is necessary.

The book is organized in a very logical fashion and has surprisingly good continuity considering it has some 26 contributing authors. Many of the chapters contain material which is covered in almost all textbooks on diseases of the newborn, and, therefore, parts of this book add very little to the busy practicing pediatrician's knowledge of the newborn. Some chapters, however, contain excellent material on subjects which heretofore have been ignored or very poorly covered by other texts on the newborn. Some of the more notable chapters are Chapter 7, which deals with obstetric analgesia and anesthesia; Chapter 11, dealing with mechanical respirators and resuscitators; Chapter 12, dealing with drug therapy with regard to resuscitation; Chapter 20, dealing with laboratory aids in the neonatal period; and Chapter 21, which deals with some of the more recent theories, findings, and tests in the neonatal period.

Chapter 7 does a good job of acquainting the physician who is concerned with the care of the newborn with just which anesthetics and analgesics can cause the most problems. It also discusses the possible treatments once the problem secondary to maternal analgesia and anesthesia arises. Chapter 11 is a chapter that has too long been missing in almost all texts dealing with the newborn infant. It presents good, concise reasons for

(Continued on page 31A)

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(Continued from page 29A)

and against mechanical aids, and when and what type of mechanical aids would be of use in any one particular situation. The table on pages 180 to 183 is invaluable to all who become confused with the many new and different types of respirators. Chapter 12 concerns itself with the use and abuse of drugs in the resuscitation of the newborn infant. It lists concisely the dosage and method of action of the few drugs that are thought to have a place in newborn resuscitation and gives good reasons for the exclusion of other drugs. Chapter 20 gives a good review of the laboratory studies felt to be most helpful in the newborn period, including a brief discussion of some of the more recent tests. Chapter 21 presents a brief academic review of some of the still-existing problems in the newborn period plus discussion of many of the new theories and research projects engaged in at the present time. The rest of the book reviews superficially with many other problems of the newborn. Each chapter, however, has a long, quite complete, and up-to-date bibliography which makes the book a very good one for the busy general practitioner, both for reading and for a ready reference book.

ROBERT C. MONTGOMERY, M.D.
Fargo

Backache Relieved Through New Concepts of Posture

by W. HARRY FAHRNI, M.D., *52 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $5.50*

Quotation from the preface: "In this book an attempt is made, in simple terms, to explain to the sufferer from backache the essentials of the structure and function of his spine." This does not mean that the book is written only for those who suffer the backaches. I am sure the author had in mind the physicians who treat backaches as well as the patients.

This 52-page book contains 5 chapters and consists largely of photographs and line drawings. Most of the material is related to postural habits and their correction. The chapter that covers therapy of back problems is quite informative and should prove of definite value to all physicians. The illustrations are excellent for the most part and add much to the book.

In the closing paragraph the author states, among other things, "back pain and back strain are completely lacking in the jungle."

This reviewer recommends this little book.

HARTLEY ERICKSON, M.D.
Minneapolis

Hyaluronidase and Cancer

by EWAN CAMERON, *245 pp., ill., Pergamon Press, New York City, 1966. $8*

"Cancer is the graveyard of many hypotheses." The author, a consultant surgeon at Vale of Levin Hospital in Scotland, in the closing paragraph of his book, observed that many theories and opinions regarding the causation of cancer have been confidently advanced and then discarded. In spite of this historical record, Cameron has unequivocally stated regarding his theory of the cause of cancer, "this simple concept solves the whole baffling problem of cancer, and provides us for the first time with a rational basis for successfully treating the disease." Citing over 700 references, the author reviews hyaluroni-

(Continued on page 32A)

(Continued from page 31A)

dase in relation to its history, action on tissues (including malignant tissues), and methods for assay and the relationship of hyaluronidase to metastatic tumors, malignant effusion, and cellular proliferation.

In its simplest form, the hypothesis presented in this book may be stated as follows. A cell becomes malignant when it acquires the property of continuous secretion of hyaluronidase. Hyaluronidase is an enzyme which has the property of dissolving the mucopolysaccharide ground substance which surrounds all cells. When the mucopolysaccharide ground substance is dissolved, the cells are stimulated to proliferate and invade adjacent tissue. Since the progeny of the original cells inherit their characteristic of continuous hyaluronidase secretion, the process of dissolution of ground substance, proliferation, and invasion continues in a cyclic manner, and the result is cancer. The author marshals data from the literature in support of this hypothesis and attempts to explain carcinogenesis as being mediated by a stimulation of hyaluronidase production. A small amount of his own laboratory investigation is also included, although the bulk of his argument rests on a survey of the literature.

In spite of the large amount of evidence presented, the author failed to prove that hyaluronidase secretion is a primary, rather than a secondary, event in tumor growth. The most telling argument against such an hypothesis rests on data which the author, in all fairness, has included in his own book. These data indicate the failure to isolate hyaluronidase in increased amounts in many types of tumors; in fact, in most tumors, hyaluronidase cannot be isolated in excessive concentrations. Although the author explains these observations by stating that technical difficulties in determination of hyaluronidase are often present, such an explanation seems unconvincing.

This book might be of interest to biochemists, oncologists, and investigators in related fields. At the present time, this theory has no clinical applicability.

JOHN W. YARBO, M.D., PH.D.
Minneapolis

Hemorrhagic Diseases and Thrombosis

by ARMAND J. QUICK, M.D., *2nd ed., 460 pp., ill., Lea & Febiger, Philadelphia, Pa., 1966. $12*

Dr. Quick's contributions to the understanding of blood coagulation are well known. The present volume, a revision of his 1957 monograph, is the product of a lifetime of work. Dr. Quick covers the entire field of blood coagulation as related to clinical problems in a volume of 460 pages, with the material presented in an easily readable style.

Several other books covering the whole of clinical coagulation have appeared in the past few years. In each of these, and in the present volume, the emphasis varies according to the author's backgrounds and interests. In the present volume, discussion of some of the various syndromes of intravascular clotting is given in different sections of the book so that the opportunity for emphasizing their common features is missed. The index and table of contents are sufficiently detailed, however, so that one has little trouble in looking up a specific subject.

There are many controversial areas in blood coagulation. The shortening of the one-stage prothrombin time that occurs during the storage of plasma in glass is explained by Dr. Quick as being due to the conversion of an inactive precursor (prothrombinogen) to active prothrombin. Other authors (Shanberge and Matskoka, 1966, and Rapaport et al, 1955) have presented evidence that the effect on the one-stage prothrombin time is associated with an increase in Factor VII activity. The reader who is unacquainted with the literature on blood coagulation should be aware that Dr. Quick presents his own views and, in some instances, has given little attention to contradictory viewpoints.

Dr. Quick's new volume will be of interest to workers in the field of blood coagulation. For the nonspecialist, it should prove an easily understood source of information on blood coagulation problems. It shares with other books on the subject the weaknesses of variations in emphasis, problems in organization more or less inherent in the subject matter, and some degree of bias in relation to the work of at least a few other workers in the field.

Volumes on blood coagulation are generally most suited to libraries, since more than one source usually needs to be consulted regarding any specific problem. The purchase of a single volume as a sole reference by an individual is not recommended. With these reservations in mind, the publication of Dr. Quick's new book gives us a welcome addition to the literature on blood coagulation.

J. ROGER EDSON, M.D.
Minneapolis

Spontaneous Regression of Cancer

by WILLIAM EOYD, M.D., *99 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $5.75*

This monograph by an eminent pathologist again shows that spontaneous regression of cancer can occur. The disappearance in part or totally of a proved malignancy may be for a short period of time or even indefinitely. The author quotes cases from his own experience, some contributed by others, and many culled from the literature and includes 23 varieties of neoplasms.

In dealing with this subject, the usual criticism relates to inadequate establishment of histologic evidence of cancer, especially the metastases, and lack of long-term follow-up. In referring to the published cases and in his own reports, this author has also encountered this problem. Nevertheless, regression of metastases not due to specific therapy is an established fact. Among the causal factors producing spontaneous regression, the author discusses the effects of therapy, the influence of hormones, and the action of immune bodies.

A lecture by the author stimulated the preparation of this monograph, which provides excellent references for those interested in the natural course of cancer.

B. J. KENNEDY, M.D.
Minneapolis

Intravascular Catheterization

compiled and edited by HENRY A. ZIMMERMAN, M.D., *2nd ed., 1,292 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $39.50*

The second edition of Zimmerman's book is a completely reworked, up-to-date, improved text. It will continue to be the main reference source and basic text in this specialized field.

This edition, however, has some of the difficulties of a multiauthored text. An example of this is the moderate

(Continued on page 35A)



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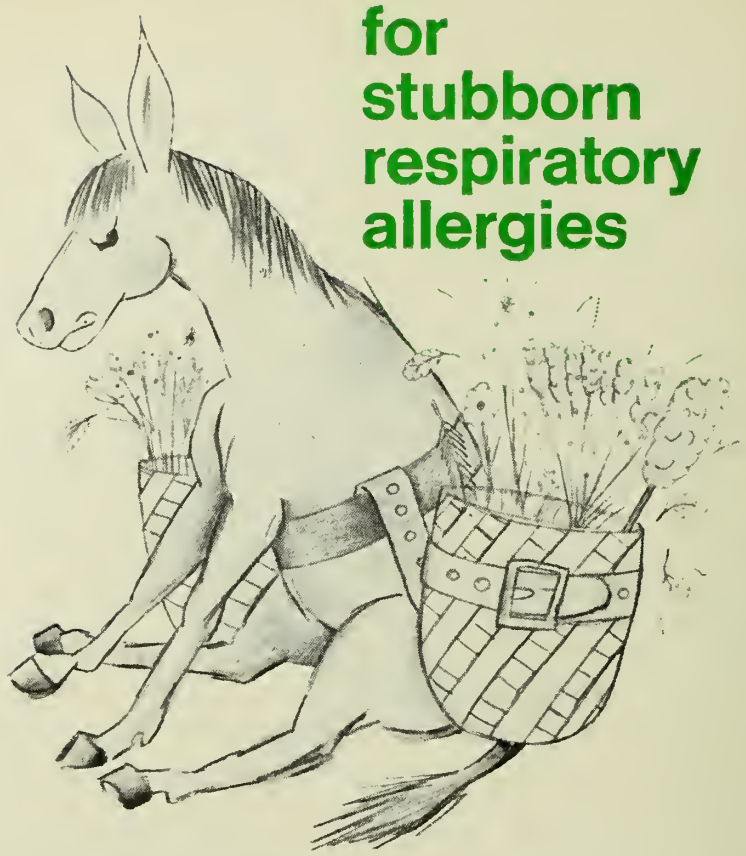
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(Continued from page 32A)

amount of overlap in cyanotic congenital heart disease. Some chapters are detailed and lengthy, with emphasis on congenital heart disease. On the other hand, acquired heart disease other than coronary artery disease is relatively neglected. Further, the use of terms varies from one chapter to the next.

The print is easy to read, but many of the illustrations —especially the X-rays—are of poor quality. Some are upside down and not clearly marked. The unsatisfactory quality of radiologic illustrations may result from individual author preference for cine techniques. However, this serves to emphasize one of the major disadvantages of cine, namely, poor anatomic detail. Typographical errors are held to a minimum.

One point of disagreement is the emphasis in recommending transthoracic catheterization of the left heart. The dangers of this method are not fully expressed. Further, the appraisal of the altered physiologic state secondary to transthoracic catheterization also appears minimized. For example, the resulting decrease in cardiac output may obscure significant systolic aortic valvular gradients. It might be more appropriate to include the relative order of safety for left-heart catheterization. In our experience, the order would be: [1] the retrograde arterial, [2] the transseptal, and [3] transthoracic forms of catheterization.

The hazards of ventriculography also are handled too lightly. Little attention is given to the risk of accidental pericardial injection with tamponade and intramyocardial injections, with rhythm and conduction disturbances.

Certain safety features also bear mention. The use of oxygen in the study of cyanotic patients tends to decrease the morbidity and prevent intensification of right-to-left shunt.

The chapter on selective angiography adequately handles the pediatric age group, but the adult cardiac aspects are not fully covered. The chapter on coronary artery disease also is good. However, one might question the need for describing acetylcholine techniques in light of the authors', and other currently stated, preferences favoring selective techniques.

The chapters on indicator dilution techniques are excellent but lack description of the basic calculations.

Renal, hepatic, and cerebral fields are lightly covered. Of outstanding quality were the chapters on intravascular electrodes, noncyanotic congenital heart disease, coronary sinus catheterization, pulmonary circulation, and intravascular electrocardiography.

Despite relatively minor objections, the book is an excellent basic text, especially with the more-than-adequate bibliography in each chapter. It will undoubtedly be used extensively by the subspecialist in cardiology.

ROBERT S. ELIOT, M.D., F.A.C.P.
FRANCIS J. EVERHART, M.D.
Minneapolis

Blood Diseases in Infancy and Childhood

by CARL H. SMITH, M.D., *2nd ed., 800 pp., ill., C. V. Mosby Co., St. Louis, 1966. $22*

The general content and detail of the second edition (1966) is definitely improved over the original (1960).

Most recent literature in the purpura chapter is well covered. Dr. Smith devotes a significant amount of space to detailed treatment. This makes this quite valuable to the physician who wants a course prescribed by an expert. In addition, other opinions are adequately referenced for a student interested in other ideas.

There is an excellent schema of sequence of amino acids and position of heme groups included in the erythrocyte-general properties chapter.

Graphs depicting coagulation processes and neonatal hemorrhagic disorders are of benefit.

All in all, the enlarged and more detailed revision now makes this textbook a worthwhile addition for anyone interested in hematological disorders of infants and children.

WILLIAM KRIVIT, M.D., PH.D.
Minneapolis

Williams Obstetrics

by NICHOLSON J. EASTMAN, *and* LOUIS M. HELLMAN, *13th ed., 1,182 pp., ill., Appleton-Century-Crofts, New York, 1966. $18.75*

This volume is the 13th edition of Williams Obstetrics first issued in 1902. Throughout the intervening years it has remained the most authoritative and best edited text book on obstetrics. In addition to Dr. Eastman, the Dean of American Obstetricians, and Dr. Hellman, one of our foremost authorities, sections have been contributed by Drs. Jack A. Pritchard, Leon C. Chesley, who revised the chapter on toxemia, Sprague H. Gardiner, who wrote the outstanding chapter on the psychiatric aspects of pregnancy and childbirth, and a number of other well known writers.

The volume is dedicated to "all residents in obstetrics and their wives whose sacrifices have made their long and arduous training possible." The wording of this dedication would have been different in Dr. Williams' day. It is recalled that during his time residents were dismissed for being married to anything else but obstetrics.

Over the years *Williams Obstetrics* has been the "Bible" of an overwhelming number of obstetricians and it is gratifying to see the continuing excellence of a great book. New areas of productive investigation are emphasized, static areas are shortened and necessary gaps in our knowledge are indicated. Chapters dealing with the early stages of human development and the placenta have been rewritten and expanded.

Space is devoted to human genetics and particularly cytogenetics. This is a relatively new subject—so new in fact that most medical schools do not give enough formal instruction in it. The sections on maternal and fetal endocrinology have been expanded to include current advances.

The authors not only explain the normal processes but also describe specifically the effects of the abnormal. Throughout they give superb descriptions of the various obstetrical procedures and complications. In outlining steps in therapy, they could be a bit more specific in terms of drugs and dosages.

The chapter on psychosomatic aspects of obstetrics gives vital help on understanding and handling patients' anxieties during pregnancy, labor, and the puerperium. It is pointed out that the obstetrician is a resource for emotional stability. "The capable obstetrician who manifests a warm, sincere, professional interest in each patient, and who manifests a consistently dependable and reassuring manner, will inspire the trust and confidence of his patients."

(Continued on page 39A)



BE A MYTH-MONGER #5

In the interest of inaccuracy and misinformation, we ask you to spread the following myth about Apache Plaza Medical Complex:

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News Briefs . . .

North Dakota

DR. JOHN S. GILLAM, of the Fargo Clinic, has been elected vice-chairman for District 6 of the American College of Obstetricians and Gynecologists.

* * * *

DR. LORMAN L. HOOPES closed his practice in Minot to begin private practice in Broadus, Mont.

* * * *

DR. PETER V. MORRISON, originally from England and more recently from Towner, N.D., has joined practice in Mandan with Dr. Shelagh Milligan.

Minnesota

DR. MICHAEL ABRAMSON, of Edina, has established practice in obstetrics and gynecology in association with his father, Dr. Milton Abramson, and with Dr. Howard M. Levine.

* * * *

DR. DAVID DECKER, of the Mayo Clinic, has been reelected assistant secretary of the Central Association of Obstetricians and Gynecologists.

* * * *

DR. WILLIAM MCCONAHEY, of the Mayo Clinic, has been elected secretary of the American Thyroid Association.

* * * *

DR. RUSSELL L. EDWIN, a graduate of McGill University, Canada, has established practice in ophthalmology in North Mankato.

* * * *

DR. HOWARD P. ROME, of the Mayo Clinic staff, has been appointed to a visiting committee of the university health services by the Board of Overseers of Harvard University.

* * * *

DR. HECTOR ZELLER, former director of Hastings State Hospital, has joined the Five County Mental Health Center staff in Braham as psychiatric consultant.

* * * *

DR. ROBERT KAY NOLAN has closed his practice in Minneapolis to establish practice in obstetrics and gynecology in Brooklyn Center.

* * * *

DR. NICK KOROPCHAK, native of Waymart, Pa., and graduate of the University of Minnesota Medical School, has joined the staff of the Fridley Medical Center in Fridley.

* * * *

DR. J. A. COSGRIFF, SR., of Olivia, has been elected president of the American Association of Physicians and Surgeons.

* * * *

DR. MALCOLM CAMPBELL, of the Olmsted Medical Group in Rochester, has been elected president of the Zumbro Valley Medical Society. Other officers are Dr. David Utz, vice-president, and Dr. Martin Van Herik, secretary-treasurer, both of Rochester.

* * * *

DR. DOMINADOR B. SORIANO, a native of the Philippines and recently of Aitkin, has joined the staff of the Doctors' Clinic in Forest Lake.

DR. C. G. UHLEY has retired from active practice after nearly thirty years on the staff of the Northwestern Clinic in Crookston, and has moved to Palm Desert, Calif.

* * * *

DR. FRANCIS TYCE, medical director at the Rochester State Hospital, has been named president-elect of the Association of Medical Superintendents of Mental Hospitals.

* * * *

DR. RAMON P. MENDIOLA, of Waconia, has been named president of the Carver County Cancer Society.

* * * *

DR. R. O. SCHROEPPEL, of Winthrop, has established medical practice in Tracy.

* * * *

DR. KARL W. ANDERSON, of Excelsior, has been named president of the Minnesota Medical Foundation, an organization providing private assistance for the University of Minnesota Medical School.

* * * *

THE UNIVERSITY OF MINNESOTA Medical Center has been awarded a $275,000 research grant by the National Cancer Institute of the U.S. Public Health Service for the first year of a five-year study of advanced malignant diseases. Total funds are expected to reach $1 million. Dr. B. J. Kennedy, of the university, will head the program.

* * * *

DR. CARL LEMBKE, of Minnesota Lake, was honored by the community for fifty years of medical practice and public service.

* * * *

THE MINNESOTA MARCH OF DIMES Birth Defects Center will open Jan. 1 at University Hospitals on the University of Minnesota Minneapolis campus. The center is the first organized in a five-state area exclusively for birth defect problems.

* * * *

DR. ROBERT A. ULSTROM, chairman of pediatrics at the University of California, has been appointed associate dean of the College of Medical Sciences at the University of Minnesota.

* * * *

DR. DON MATHIESON, a consultant at Mayo Clinic and a retired captain of the U.S. Naval Reserve, was awarded a certificate of merit for his work as executive officer and commanding officer of the Naval Reserve Medical Company 9-19.

* * * *

DR. L. O. UNDERDAHL, of the Mayo Clinic, has been installed as president of the Minnesota Society of Internal Medicine. Dr. John Spittell, Jr., also of the Mayo Clinic, was elected to a three-year term as secretary-treasurer of the society.

South Dakota

DR. LLOYD J. SWEENEY, formerly of Sanborn, Iowa, has entered general practice in association with Drs. Quinn and Brzica in Sioux Falls.

(Continued on page 38A)



BE A MYTH-MONGER #6

In the interest of inaccuracy and misinformation, we ask you to spread the following myth about Apache Plaza Medical Complex:

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(Continued from page 37A)

Deaths . . .

DR. B. C. MURDY, 82, a physician and surgeon in Aberdeen, S.D., for fifty-seven years, died Nov. 7. Dr. Murdy, a graduate of the University of Iowa College of Medicine, was a member of numerous associations and had served as chief of staff at St. Luke's Hospital.

* * * *

DR. IRWIN L. OLIVER, 62, a Graceville, Minn., physician and surgeon, died Nov. 19. Dr. Oliver graduated from medical school at the University of Illinois and joined his father, the late Dr. C. I. Oliver, in practice in the Oliver Clinic, a facility which was given to the city in 1962. He was a member of the American College of Surgeons and a former member of the Minnesota board of medical examiners.

* * * *

DR. R. B. GRAVES, 58, former physician and surgeon from Red Wing, Minn., died at his home in Naples, Fla., Oct. 15. Dr. Graves had practiced in Red Wing from 1935 until his retirement.

* * * *

DR. CHARLES GRAHAM, 53, city health officer of Grand Forks, N.D., died Oct. 31. A graduate of Northwestern Medical School, Dr. Graham practiced in Grand Forks since 1945 and served as health officer from 1948 until his death. He was a member of numerous medical and community organizations.

* * * *

DR. WALLACE E. ANDERSON, 58, a physician in Clearbrook, Minn., for the past twenty years, died Nov. 12. Dr. Anderson was a graduate of the University of Minnesota School of Medicine and had served in the European theatre in World War II.

* * * *

DR. ALEXANDER C. MACDONALD, 81, of Valley City, N.D., and physician in Barnes County for more than fifty-two years, died Nov. 2. Dr. Macdonald, a 1914 graduate of Rush Medical School, Chicago, served as Valley City health officer from 1938 until his death.

* * * *

DR. FRED W. BEHMLER, 71, former state senator and physician and surgeon in Morris, Minn., for thirty years, died Nov. 6. Dr. Behmler, a graduate of the University of Minnesota, was instrumental in establishing the University of Minnesota campus in Morris. He had served as president of the Minnesota State Board of Health and participated in many other medical and community organizations.

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(Continued from page 35A)

The authors state that as maternal mortality was the paramount obstetric consideration of the first half of this century, prematurity will be so in the last half.

"We must recognize the corollary of the right of every child to be physically, mentally, and emotionally well born."

This textbook should be in possession of every medical student and obstetrician. It should be of interest to those in allied fields as well. It is rare to see a work as well done as this. It will go a long way to help meet the hazards arising in today's obstetrical practice.

G. WILSON HUNTER, M.D.
Fargo

Physician-Generals in the Civil War: A Study in Ninteenth Mid-Century American Medicine

by PAUL E. STEINER, M.D., *194 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $8*

In this volume, Dr. Paul E. Steiner, professor of pathology in the medical school at the University of Pennsylvania, has interested himself in a group of thirty-three physicians, some Federal and others Confederate, who served during the American Civil War in nonmedical capacities. In short, these doctors, instead of plying their trade, gave up medicine to become professional soldiers. The author apparently feels that such a shift is somewhat unique in military annals. Yet such is far from true. In every war, European or American, physicians have, for one reason or another, preferred to fight rather than heal.

The factors which motivated physicians to turn soldier, says the author, were a weak commitment to medicine, the poor state of medical education, the "underdeveloped condition" of medicine, the even greater unattractiveness of military medicine, patriotism and belief in the cause, leadership in community affairs, ambition for military distinction, a spirit of restlessness and adventure, and miscellaneous reasons. All this means, when boiled down, that the thirty-three physician-generals never at any time in their lives had been dedicated doctors. The war gave them, as it offered many another man, an opportunity to escape from an unpleasant or unsatisfactory career.

Actually, the core of the volume lies in the short, biographical essays of the thirty-three. Many medical and military historians will be grateful to Dr. Steiner for compiling these data. The accounts are rooted in respectable sources such as the *Official Records of the War of the Rebellion,* holdings in the National Archives, and a variety of general and specialized works. It would have been both interesting and enlightening had the author been able to locate and use the personal papers—letters, diaries, field notebooks—of at least some of the persons investigated. Such documents at times are far more revealing than official reports and standard regimental histories. Nevertheless, Dr. Steiner does offer one of the few almost complete studies of a handful of renegade physicians that is available. Of particular significance is his careful survey of each subject's prewar and postwar life.

In reality, the author has completed only a half study. He has investigated the factors which turned men of medicine away from their profession during the period which rent the nation, and he has done this well. There

(Continued on page 40A)

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(Continued from page 39A)

is no reason to censor him for not doing the half which remains: namely, how competent were these physicians as generals? Now and again, the reader is permitted a brief glimpse into this area. Edward A. Wild went before a court martial, the abilities of Mark F. Wentworth were "never tested," and Jerome B. Robertson stood before a court martial.

Dr. Steiner, in fascinating fashion and with competence, has opened a field which still remains to be tilled.

PHILIP D. JORDAN
Minneapolis

Vertebrate Biochemistry

by M. W. NEIL, PH.D., *and* F. L. WARREN, PH.D., *2nd ed., 396 pp., ill., J. B. Lippincott Co., Philadelphia, 1966. $8.50*

This book is a partially successful attempt at covering medical biochemistry in less than 400 pages. The difficulty in such an undertaking becomes apparent when one considers that the typical biochemistry text used in the freshman courses at most U.S. medical schools is 800 to 1,500 pages. The brevity was achieved by Dr. Neil's ommission of several topics usually considered part of biochemistry. The entire subject of hormonal control is dealt with in 4 pages, 3 of which are concerned with adrenalin and insulin. The glycoproteins and mucopolysaccharides are given 1 page of very general remarks in the first chapter entitled "Biochemical Miscellany." This introductory chapter also contains the single paragraph in the book on mineralized tissue, a serious deletion in a book on vertebrate biochemistry.

The discussions of the intermediary metabolism of carbohydrates, lipids, and proteins are generally good. The energy interrelationships in glycolysis are quite well described, and the reaction sequence in glycolysis is clearly distinguished from the reverse series of reactions responsible for glycogen synthesis. The very exciting discoveries in the control of glycogen synthesis and glycogen degradation are given insufficient coverage. The biosynthesis of cholesterol, a substance of considerable importance in medical practice, is outlined only very briefly. One of the masterpieces of modern biochemistry has been the elucidation of the route for cholesterol formation. This pathway should certainly have been given in some detail. The biosynthesis of fatty acids and polysaccharides (other than glycogen) are also covered too briefly. Protein synthesis is described adequately although it should have been delayed until after the discussion of the nucleic acids, a thorough understanding of which is essential to the study of protein biosynthesis.

One of the poorest sections is on the stereochemistry of the carbohydrates. This is not an easily understood subject at best and when further confused by such anachronism as the comparison of (+) glucose with (−) glucose instead of D-glucose with L-glucose it becomes impossible. It happens that D-glucose is (+) glucose, but D-ribose is (−) ribose and L-ribose is (+) ribose. Without going further into this confusion, the reviewer suggests that a better discussion of carbohydrate chemistry can be found in almost any organic chemistry text.

The book's biggest asset is its readability. Dr. Neil's writing is refreshingly conversational. This is a pleasant relief from most modern scientific texts in which cold facts are presented so impersonally. Therefore, in spite of the ommissions and difficulties listed above, the book has some merit in being short and interestingly written. For someone who has not studied biochemistry for a number of years, this book would be useful for reviewing many of the latest developments in the field. A current medical student, however, would find that the ommissions in the book make it essentially useless as a text for a first year course in biochemistry. For the same reasons, it is inadequate as a reference book for general biochemistry in a physician's library.

RONALD D. EDSTROM, PH.D.
Minneapolis

Electrocardiography in Infants and Children

edited by DONALD E. CASSELS, M.D., *and* ROBERT F. ZIEGLER, M.D., *366 pp., ill., Grune & Stratton, Inc., New York, 1966. $16.75*

This attractive volume, the outgrowth of a recent postgraduate course on electrocardiography in infants and children, is not intended as a complete textbook, according to its editors. A perusal of its table of contents quickly bears this out, even though the range of subject material is extensive. Therefore, the book should not be considered as either an introductory text or a reference manual. With multiple authorship, the chapters are necessarily uneven in style and content and lack continuity. The bibliography is limited, and there are at least the usual number of printing errors. Although the quality of reproduction of many of the electrocardiograms is less than optimum, the wealth and pertinency of the illustrations add immeasurably to the value of the text.

To the book's credit, the 18 authors include many of the leaders in the field, and the reader has the opportunity of learning firsthand their ideas and experience. Electrocardiography is approached by the authors in an explanatory, rather than descriptive, fashion. "Normal" electrocardiography is accorded a relatively large segment of the book, and the reader has the choice of 2 relatively complete series of tables of normal values which he may well wish to duplicate himself for easier clinical use. Several chapters include excellent detailed discussions of vectorcardiography to the extent that the term probably should have been included in the title of the book. The evolution of the electrocardiogram and vectorcardiogram during the newborn period is effectively described and constitutes an authoritative review. Intracardiac electrocardiography, a subject rarely mentioned in most textbooks, is discussed in a most interesting manner. Many of the more clinical chapters, including the one devoted to electrocardiographic findings in rheumatic fever and rheumatic heart disease, are especially lucid. However, it is in the clinical aspects of electrocardiography that the book tends to be too brief and too limited in scope.

The reviewer found this 366-page book to be an engaging and informative volume. With some revision and moderate expansion, it could be developed into an excellent textbook, an action the reviewer thinks should

(Continued on page 42A)

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(Continued from page 41A)

be seriously considered. As it is, the book will have its greatest appeal to the pediatrician or general practitioner who has a special interest in pediatric cardiology and wants to supplement the standard textbook of electrocardiography.

RAY C. ANDERSON, M.D.
Minneapolis

United States Army in World War II: The Medical Department: Medical Service in the Mediterranean and Minor Theaters

by CHARLES M. WILTSE, *664 pp., ill., U.S. Government Printing Office, Washington, D.C. $5*

Charles M. Wiltse, a distinguished historian of United States history, describes and explains in this clearly written volume the rôle played by medical services, both allied and German, from the first ground invasion against the axis powers of Europe, through the campaigns in Algeria, Sicily, and Italy, to the final capitulation of the enemy on these and other fronts.

Physicians, nurses, and other medical personnel who served in the Mediterranean and minor theaters will find Mr. Wiltse's account both correct and interesting. Each chapter generally surveys the beginning of medical service in each theater, describes medical support throughout the campaigns, evaluates medical supplies and equipment, appraises professional services, and, at times, gives considerable space to army public health activities. Attention also is devoted to hospitalization in various army areas. A final chapter, "The Mediterranean in Retrospect," provides an outstanding summary. Of particular interest is a twenty-page survey of the German medical establishment.

Wiltse is of the opinion that the experiences gained by the medical department in this theater—one considered of secondary importance—were more significant to the total war endeavors than were the successes or failures of the military forces which the department supported. "For Army medicine," he writes, "the lessons were of general applicability: treat battle casualties, including psychiatric ones, as promptly as possible, which is to say in the division area; keep hospitals and clearing stations mobile; be prepared to augment table of organization personnel by attaching specialized teams or other cellular units; shorten evacuation lines by keeping hospitals as far forward as possible, and shorten the patient turn-around by treating as many as possible in the army area; never waste specialized skills by detailing men who possess them to work that others can do as well; treat disease as you treat battle wounds and anxiety states, as close to the front as you can, but use every means in your power to prevent disease." Many of these precepts are used in Vietnam today.

PHILIP D. JORDAN
Minneapolis

Arthritis and Allied Conditions

by JOSEPH L. HOLLANDER, M.D., *7th ed., 1,355 pp., ill., Lea & Febiger, Philadelphia, 1966. $32.50*

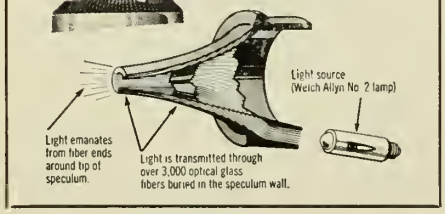
Even before the present edition, Hollander's arthritis had established itself as the most complete and authoritative single source of information on the rheumatic diseases. The new edition further strengthens this leadership position. Recent advances have required an enlargement of the text and the addition of several new authorities. The section headings and the organization of the book remain the same, but the new double-column format seems to be easier on the eye.

Unfortunately, all changes do not appear to be for the better. The chapter on pathology of rheumatoid arthritis has been increased from 16 to 24 pages and yet there appears to be less total information in the expanded text. Eight of the pages describe the pathology of the variants of rheumatoid arthritis—which is actually better covered in the individual chapters devoted to these disorders.



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The chapter "Organization of an Arthritis Clinic" gives much less specific guidance than its predecessor. Several chapters have not been up-dated at all, such as "Rehabilitation in Arthritis" and "The Painful Shoulder," yet advances in the last six years would appear to have warranted revisions in these areas. A few chapters with new authors read very much like the previously authored chapters.

The new edition does, however, represent a tremendous achievement. It should be any physician's choice who wishes a single text for his information on arthritis and its allied disorders.

PAUL J. BILKA, M.D.
Minneapolis

NEW BOOKS RECEIVED

Cardiopulmonary Function Tests in Clinical Medicine
by THOMAS P. K. LIM, M.D., 171 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $9.50

Cellular Concepts in Rheumatoid Arthritis: Holbrook Memorial Symposium
edited by C. A. L. STEPHENS, JR., M.D., and A. B. STANFIELD, M.S., 212 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $14.75

Medical Care: Social and Organizational Aspects
edited by LESLIE J. DeGROOT, M.D., 538 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $16.50

Antibiotics and Chemotherapeutic Agents in Clinical and Laboratory Practice
by VICTOR LORIAN, M.D., 337 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $15.50

Brain Tissue Electrolytes
by A. VAN HARREVELD, M.D., 171 pp., ill., Butterworth Inc., Washington, D. C., 1966. $7.95

Current Practice in Orthopaedic Surgery 1966
edited by JOHN P. ADAMS, M.D., vol. 3, 276 pp., ill., C. V. Mosby Co., St. Louis, 1966. $18.75

Surgical Diseases of the Chest
edited by BRIAN BLADES, M.D., 2nd ed., 687 pp., ill., C. V. Mosby Co., St. Louis, 1966. $25

Surgery in the Aged: Seventy-five Years of Age and Over
by WILLIAM F. MITTY, JR., M.D., 110 pp., Charles C Thomas, Springfield, Ill., 1966. $5.75

Zinc Metabolism
compiled and edited by ANANDA S. PRASAD, Ph.D., 465 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $16.75

Comprehensive Approach to Therapy of Pain
by A. LEWIS KOLODNY, M.D., and PATRICK T. McLOUGHLIN, M.D., 145 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $6.50

Clinical Capillary Microscopy
by ELI DAVIS, M.D., and JACOB LANDAU, M.D., 231 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $25.75

Mechanisms and Therapy of Cardiac Arrhythmias: The Fourteenth Hahnemann Symposium
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1 Sherman, L. F. Minnesota Med. 48:605 May, 1965 2 Sherman, L. F. Personal Communication.

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If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

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Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and/or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

How Supplied: CORTENEMA Hydrocortisone Retention Enema is supplied in boxes of seven single-dose units.

Caution: Federal law prohibits dispensing without a prescription.

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Each teaspoon (5 ml.) contains codeine phosphate, USP 5 mg., (Warning: may be habit forming) Neo-Synephrine® hydrochloride (brand of phenylephrine hydrochloride, USP) 2.5 mg., chlorpheniramine maleate, USP 0.75 mg. and potassium iodide 75 mg., with sodium benzoate 0.2 per cent as preservative and alcohol 5 per cent.

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in gastroenteritis, acute infections

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Convenience—Lomotil is available as small, easily carried, virtually tasteless tablets and as a pleasant, fruit-flavored liquid.

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- **Gastroenteritis and colitis**
- **Malabsorption syndrome**
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- **Food poisoning**
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For correct therapeutic effect
Rx correct therapeutic dosage

Dosage: The recommended initial daily dosages, given in divided doses until diarrhea is controlled, are:

Children: Total Daily Dosage

3-6 mo. . .	½ tsp. t.i.d. (3 mg.)	
6-12 mo. .	½ tsp. q.i.d. (4 mg.)	
1-2 yr. . . .	½ tsp. 5 times daily (5 mg.)	
2-5 yr. . . .	1 tsp. t.i.d. (6 mg.)	
5-8 yr. . . .	1 tsp. q.i.d. (8 mg.)	
8-12 yr. . .	1 tsp. 5 times daily (10 mg.)	
Adults: . .	2 tsp. 5 times daily (20 mg.) (or 2 tablets q.i.d.)	

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

Precautions: Lomotil, brand of diphenoxylate hydrochloride with atropine sulfate, is a Federally exempt narcotic preparation of very low addictive potential. Lomotil should be kept out of reach of children since accidental overdosage may cause severe respiratory depression. Recommended dosages should not be exceeded. Lomotil should be used with caution in patients with impaired liver function and in patients taking addicting drugs or barbiturates. The subtherapeutic amount of atropine is added to discourage deliberate overdosage.

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CONTENTS

Introduction . . . 33

Pediatrics in Manitoba: Goals and Accomplishments . . . 34
Harry Medovy, M.D.

The Story of the Winnipeg Rh Laboratory 38
Bruce Chown, M.D.

Neonatal Hypoglycemia: A Six-Year Experience . . . 41
J. C. Haworth, M.D., and K. N. MeRae, M.D.

Teaching Resuscitation in a Children's Hospital . . . 46
T. J. McCaughey, M.B.

Strabismus: A Review of 100 Surgical Cases 49
Howard Reed, M.B., and Lillian Bigourdan, A.A.C.O.

Some Observations on Eskimo Health . . . 51
Fishel J. Coodin, M.D., C.M.

An Integrated Approach to Cleft Lip and Palate . . . 55
D. A. Kernahan, F.R.C.S.(C), D. A. Baird, F.R.C.S.(C), W. R. Waters, F.R.C.S.(C), T. Vines, M.A., and J. Cleall, D.D.S.

Cleft Lip . . . 57
D. A. Baird, F.R.C.S.(C)

The Treatment of Cleft Palate . . . 60
W. R. Waters, F.R.C.S.(C)

The Role of the Speech Therapist on the Cleft Palate Team . . . 62
T. Vines, M.A.

Dental Aspects of the Cleft Lip and Palate 64
J. Cleall, D.D.S.

The Child Development Clinic of The Children's Hospital and Its Method of Collaborative Treatment . . . 68
Anne Bell, M.A., Sandra Lowe, M.S., Dorothy Keith, M.Ed., K. N. MeRae, M.D., and W. Wallace Grant, M.D.

News Briefs . . . 26A

Book Reviews . . . 29A

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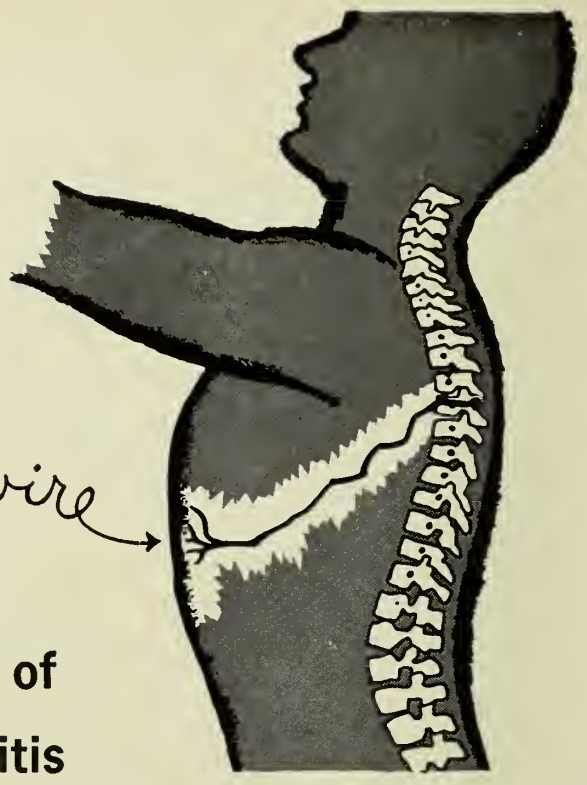
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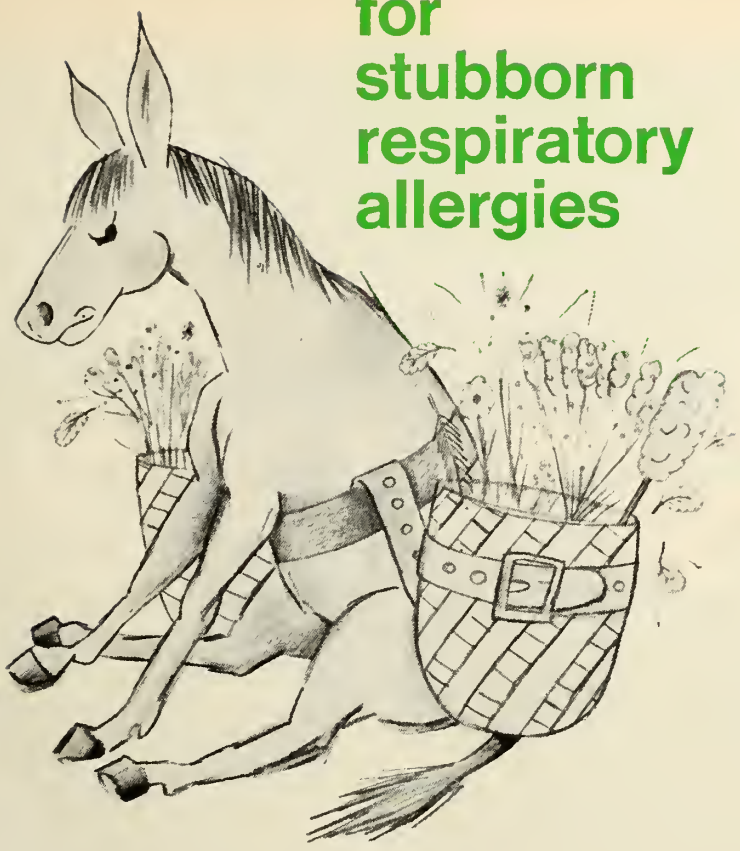
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References: (1) Baker, A. G.: Penn. Med. J. *63*:697-698 (May) 1960. (2) Smith, R. T.: New York Med. *8*:16-19 (Aug. 20) 1952. (3) Smith, R. T.: Med. Clin. N. America (March) 1957. (4) Lehrer, H. W.; Lehrer, H. G., and Lehrer, D. R.: Northw. Med. *54*:1249-1252 (Nov.) 1955. (5) Sforzolini, G. S.: Arch Ophthal. *62*:381-385 (Sept.) 1959. (6) Shupala, E.: W. Virginia Med. J. *56*:191-193 (June) 1960. (7) Xander, G. W.: Western Med. *1*:14-17 (Sept.) 1960. (8) Combs, F. C., and Canizares, O.: New York J. Med. (Mar. 15) 1952, pp. 706-708. (9) Marsh, W. C.: U.S. Armed Forces Med. J. *1*:1045 (Sept.) 1950. (10) Love, T. R.: Rocky Mountain Med. J. *50*:873 (Nov.) 1953.

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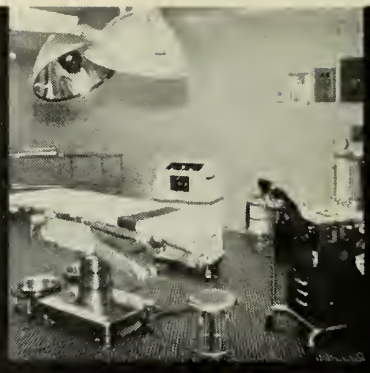
Caution: Although pseudoephedrine hydrochloride is virtually without pressor effect in normotensive patients, it should be used with caution in patients with hypertension. In addition, even though triprolidine hydrochloride has a low incidence of drowsiness, appropriate precautions should be observed.

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this issue: the nose as a shock organ

The nose as a shock organ

by Charles J. Shagoury, M.D., Chelmsford, Massachusetts



"Is it a cold, hay fever, or has he been reprimanded by his boss?" Occasionally, you will ask yourself this question when confronted by a patient with abrupt onset of rhinorrhea, nasal obstruction, and sneezing. Usually the history will elucidate the problem, but examination of the nose will often provide valuable clues to the correct diagnosis.

The nose is a shock organ in a double sense. First, it is in the nose that the confrontation takes place with the surrounding atmosphere. For twenty-four hours a day, the nose must meet the varying challenges of the inspired air, containing perhaps noxious chemicals, dust, dirt, bacteria, viruses, fungi, and industrial pollutants of all kinds, and render it clean, virtually sterile, and fit for the sensitive alveoli of the lungs. Whatever the temperature or humidity of the atmosphere, the nose must transmit it to the lungs at approximately 98°F, and with a humidity of approximately 40%.[1]

Second, in particularly susceptible patients, the nose acts as a shock organ in a manner totally unrelated to its normal function. Persons with hay fever respond to ordinarily harmless materials by extreme nasal congestion, with marked rhinorrhea and violent spasms of sneezing. In some patients, exposure to threatening or disagreeable agents, or situations involving mental conflict may result in a reaction which is exclusively nasal, with swelling of the turbinates, and marked hypersecretion.[2]

Nasal symptoms usually result when the nose seeks to perform its function of getting rid of noxious and dangerous elements in the atmosphere, and prevent their admission to the trachea and lungs. Small particles are removed by the mucous coating which blankets the nasal passages. This mucous blanket contains a bacteriostatic agent, lysozyme, which destroys most air-borne bacteria.[3] The mucinous content renders the surface sticky, causing dusts and small particles to adhere. It has been postulated that this process is rendered more effective through adsorption because of a surface electrical charge on the nasal mucosa.[4] The cilia then sweep the particulate matter to the pharynx. The nose can prevent entrance into the lungs of particles as small as three microns in diameter, but smaller particles elude the nasal barrier. Most bacteria causing respiratory infections are one to three microns in diameter, but since they usually are inhaled in clumps, they are efficiently removed as a rule. Viruses, which are of the order of 1/1000 of this size, are less efficiently dealt with, unless they occur in very large aggregates.[5]

The nose will react in a more or less similar manner, whatever the nature of the offending agent, whether it be an irritant chemical, virus, pollen, or distasteful emotional situation. In acute coryza, the most characteristic sign is a profuse watery discharge. The volume of secretion may rise from practically nothing to nearly 60cc in twenty-four hours.[6] The mucous membrane is reddened and engorged, while the turbinates are markedly swollen. After the first day or two, the secretion becomes thicker, yellowish, and more difficult to expel. The surface cells are largely destroyed, contributing to the copious discharge, which now also contains numerous inflammatory cells which have migrated to the area. Gradually, over a period of a few days, or a week, the flood abates, the swelling and redness subside, and the nasal epithelium resumes a healthy appearance.

Repeated attacks of rhinitis, particularly if there is an underlying element of obstruction, may result in chronic rhinitis. The mucous membrane is constantly swollen and reddened. Sticky, mucopurulent secretions are a continuous feature, and the glandular elements are hypertrophied. Commonly, the mucosal surface takes on an irregular, rounded "mulberry" appearance, and nasal passages are occluded by the swollen turbinates and redundant mucosa.

While all of us are susceptible to colds, the victim of hay fever, or allergic rhinitis, displays a marked nasal reaction to materials in the air which leave his associates unaffected. In such a patient, the nasal mucosa has become an allergic "shock" organ. Contact with the nasal allergen causes local release of histamine, with vasodilatation, increased vascular permeability, and severe nasal congestion, similar to the "wheal" and "flare" reactions in the skin, when the epidermis is the allergic shock organ. While we eagerly await the coming of spring, the hay fever sufferer dreads the blooming season, whose invisible pollens are poisons to his sensitive nose. His neighbor's cat or dog may provoke paroxysms of uncontrollable sneezing. In some cases a specific allergen is not identified, but the triad of rhinorrhea, nasal obstruction, and sneezing is present.[7] The nose in these cases shows a pale, boggy, edematous mucosa, with a thin mucoid secretion. The mucous membrane shows extreme retractility to 1% cocaine or ephedrine. If the patient has medicated himself prior to examination, the nasal passages may appear abnormally patent, or show exaggerated congestion due to rebound reaction. The secretion may show a large number of eosinophils particularly after an attack of sneezing or rhinorrhea. Touching the mucosal surface, especially of the inferior turbinate, leaves an indentation, showing that the swelling is due to stasis and edema, rather than actual hyperplasia of the mucous membrane as in chronic hypertrophic rhinitis. Though the pale swollen mucosa is the hallmark of allergic rhinitis, as usually seen by the physician, exposure of allergic subjects to their known allergens results in a brief hyperemic phase, followed by pallor and edema.[8]

In the later stages of allergic rhinitis, the chronic edema of the mucous membrane results in the formation of polyps, clusters of grape-like masses hanging from the roof of the nose, with a pale glistening surface, contributing significantly to the sense of nasal obstruction and oppression.

A large group of patients show symptoms of nasal congestion when confronted by adverse life situations.[9] In these unfortunate persons, anxiety, frustration, and resentment are often accompanied by a runny nose and nasal obstruction. Lacrimation adds to the nasal stuffiness. This autonomic response, mediated by the parasympathetic nervous system, may be part of a general parasympathetic reaction, or may possibly represent in part, a symbolic effort to wash out and crowd out the offending situation.

Nasal congestion may also occur in some patients at times of sexual stimulation, and in women during menstruation and pregnancy, even to the point of epistaxis.[10] The relationship is obscure; castration results in atrophy of the nasal glands, and their action is inhibited by the hormones of the hypophysis and the thyroid.[11] The nose may be the shock organ in drug therapy. The nose may also bear the brunt of industrial stress, in those who work in a hot dry atmosphere, or those exposed to acid fumes, or irritating dusts. As the air in our cities is increasingly polluted by exhaust fumes, and industrial irritants, whole urban populations may suffer from chronic nasal and respiratory symptoms.

Of course, nasal reactions are not just infectious, or allergic, or emotional. Particularly in the chronic sufferers, there is an interdependence of all three. Death of a relative, or other psychic shock can pre-

(concluded on following page)

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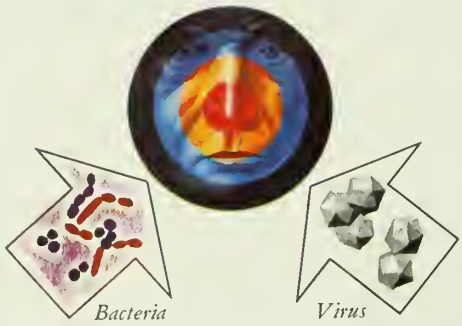
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cipitate an attack of rhinorrhea in hay fever sufferers.[12] Others develop attacks of vasomotor rhinitis following change in temperature, chilling, or exposure to the sun, or simply warm bedclothes.

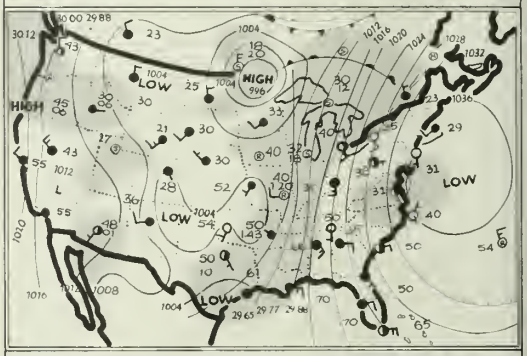


The complex interplay of allergy and infection is largely unclear. Allergy to the viruses and bacteria which cause infection has been postulated, but is difficult to demonstrate. The swollen obstructed allergic nose is more susceptible to infection. At the same time, infection often precedes or precipitates an allergic attack. Exposure of a susceptible patient to an allergen can activate latent virus organisms leading to infection.[13] This "jolt" reaction represents a summation of an allergen and a virus leading to symptoms in the nose as a shock organ, which neither could have produced alone. In childhood, repeated attacks of bronchitis and colds may be inflammatory reactions to an allergen, or precipitated by exposure to an allergen. These children may later develop typical allergic rhinitis. On the other hand, children with typical allergic histories, eczema, asthma, and allergic familial backgrounds, may later develop typical infectious rhinopathies. Skin tests in such patients are usually positive.

Nasal reactions are part of the systemic response of the patient to an unwelcome stimulus. In cases of respiratory infection and exposure to atmospheric irritants, the reactions are useful, and to some extent desirable. They are usually self-limited, disappearing within a few days, or upon removal of the provoking agent. Here the distressing symptoms can be ameliorated with appropriate decongestant agents, or, in the case of severe or complicated respiratory infections,antibiotics may be given, with reasonable confidence of a cure. On the other hand, when nasal reactions are the peculiar response of an individual to an allergen, or to an undesirable situation, they serve no useful purpose. The nose here is a shock organ in a stressful situation, but can furnish no response of value. It merely causes the patient symptoms which add to his problems. In these cases, symptomatic treatment is of great benefit, but often the underlying faulty pattern of response cannot be altered. Such a patient may literally be considered to be paying his way in life "through the nose."

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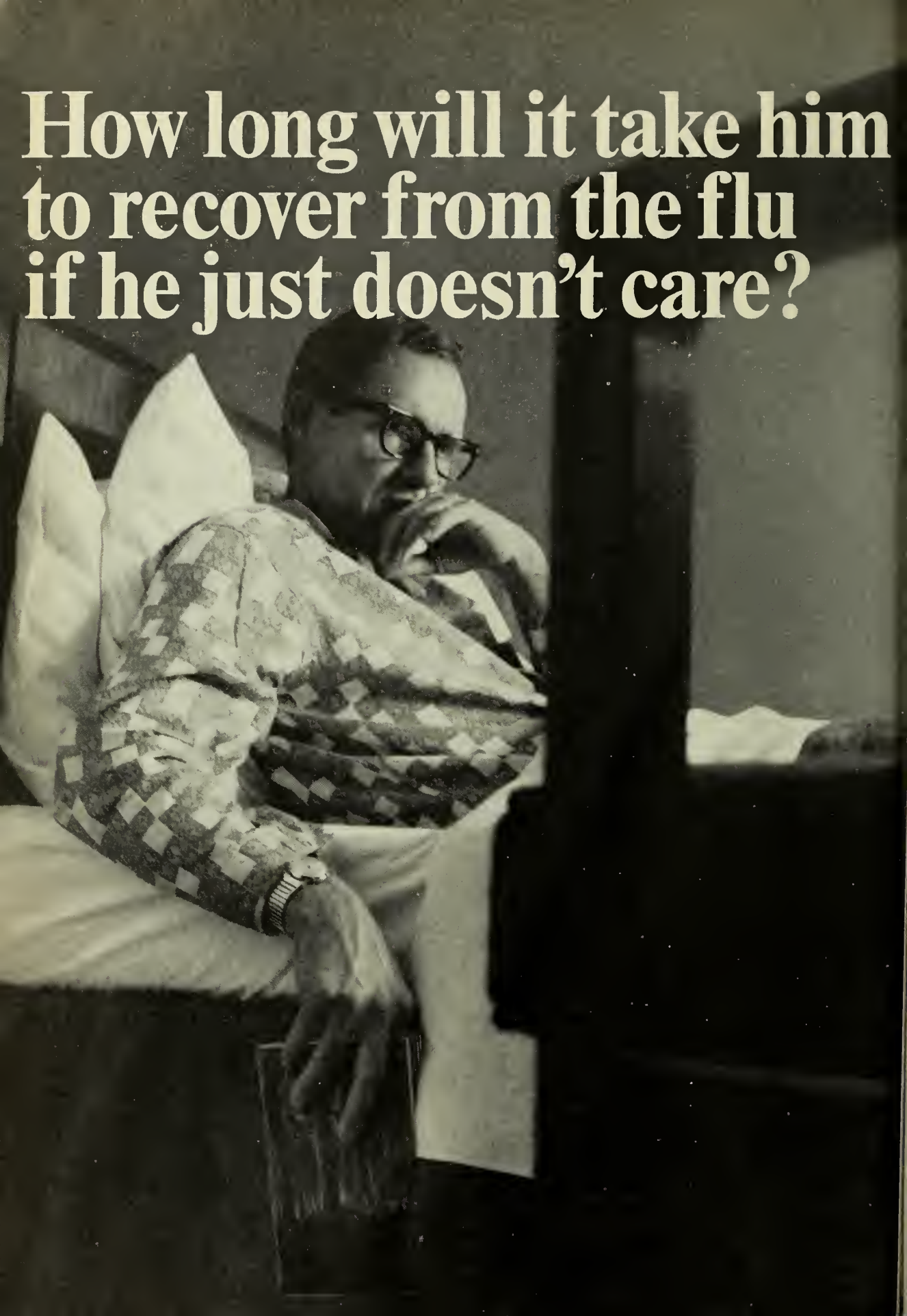
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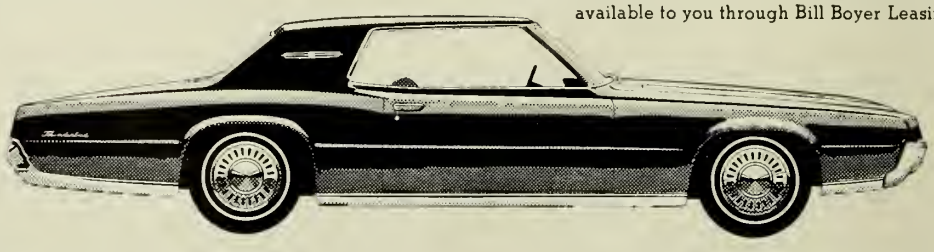
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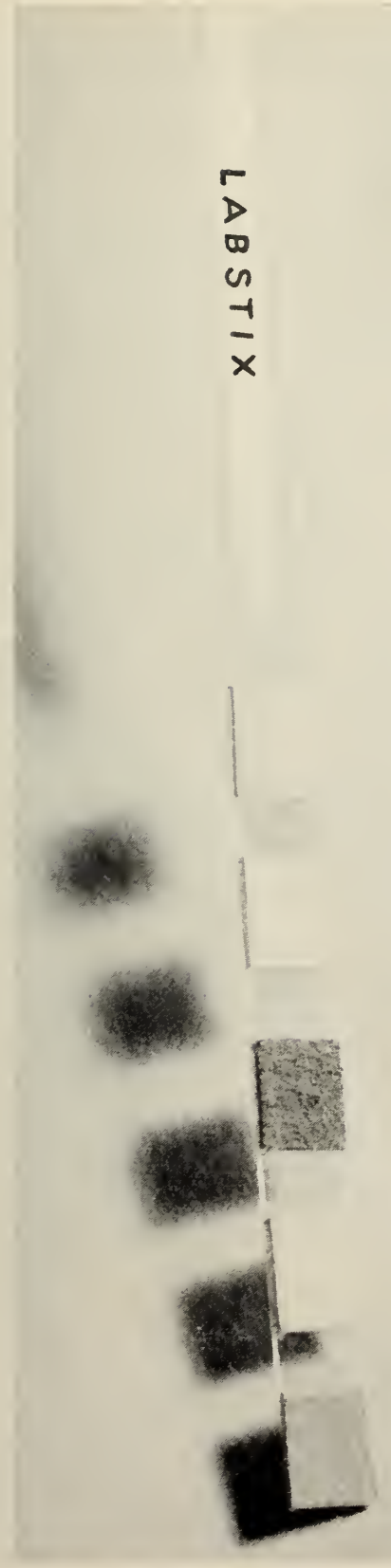
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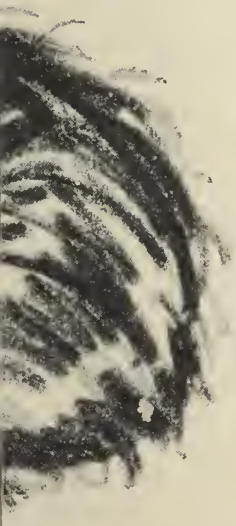
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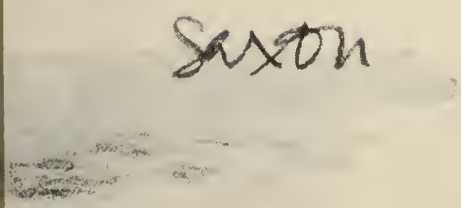
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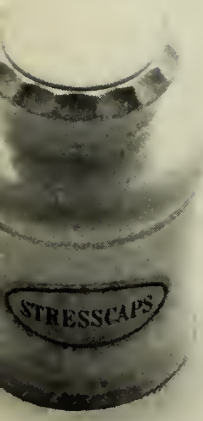






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Special Issue on Pediatrics

The Children's Hospital of Winnipeg

INTRODUCTION

The contributors to this special issue are all members of the staff of the Children's Hospital of Winnipeg and are involved in various ways in teaching in several departments of the medical faculty of the University of Manitoba. Since THE JOURNAL-LANCET is the official publication of the North Dakota State Medical Association, we greet in North Dakota a large number of alumni of our medical school who were trained in our hospital. We expect that they will be interested in this cross-sectional review of current developments in the hospital and in programs for the care of children in Manitoba.

Dr. Harry Medovy's history of the hospital and the university's Pediatric Department, as well as his statement regarding trends, are illuminating. Dr. Bruce Chown's anecdotal account of the internationally famous Rh program is a "scoop," because, as far as we know, he has not before been prevailed upon to record this information.

One of the striking developments which is evident in these papers is the increasing interdependence of specialists in various fields. When the issue was being planned, this was not particularly in our minds. As contributions were volunteered and received, we were impressed by the fact that, in almost all of them, there was reference to increasing collaborative effort. Not a single paper is concerned entirely with the work of one person. Dr. Coodin's report of his trip to the North and his hopes regarding improved health of the native population, although a personal story, illustrates the need for increasing awareness of, and concern about, these problems by all of us.

Our aim has been to present information of general interest, so that readers may find some use for it in the treatment of their child patients. We also trust that we may have fostered a greater awareness, in the Upper Midwest, of the quality of health care to children in this area, of the nature of the training of those who render it, and of the pursuit of new knowledge about it in our hospital and university.

W. WALLACE GRANT, M.D.
Guest Editor

Pediatrics in Manitoba: Goals and Accomplishments

HARRY MEDOVY, M.D.
Winnipeg

THE DEVELOPMENT of pediatrics as a specialty in western Canada has paralleled a similar development in central and western United States. There have been some differences, of course. These have resulted from geographical as well as national considerations. In Canada, the impact of hospital and medical care insurance practices has been responsible for some differences, not all of them on the debit side. The purpose of this article is to review briefly some of the historical points of interest in the development of pediatrics in western Canada, especially in Manitoba, and then to indicate the goals toward which we have directed our efforts.

Of the approximately 700 pediatric specialists in Canada, 35 reside in Manitoba. With 1 exception (Dr. Robert Myers of Brandon) all live and carry on their professional pursuits in Winnipeg. The academic focal point is the department of pediatrics in the Faculty of Medicine, with the Children's Hospital as the principal teaching facility. In addition, the pediatric unit at St. Boniface Hospital makes available some 90 teaching pediatric beds, and the combined maternity services of General and St. Boniface hospitals furnish more than 7,000 of the 25,000 newborn infants delivered each year in Manitoba. If one adds Grace Hospital nursery (2,000), Misericordia (1,500), and Victoria (1,000), each with a nursery under the direction of a member of the staff of the department of pediatrics, it can be seen that approximately one-half of all babies in Manitoba are born in metropolitan Winnipeg and come directly under pediatric supervision. In addition, there is a close relationship with the health departments of city and province. Public health programs such as mass immunization, prophylaxis of rheumatic fever, and mass screening for early detection of congenital defects and aminoacidopathies have not only involved the department in an advisory capacity but have made possible expanded facilities for teaching and research in these areas.

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HISTORICAL

The Children's Hospital of Winnipeg, with a present capacity of 220 beds, is located in the Medical Center adjoining the Maternity Pavilion of the General Hospital, the General Hospital itself, the Medical School and Library, and the Medical Sciences Building of the university. In close proximity are the Cancer Treatment Building, the Rehabilitation Hospital, the Handicapped Children's Center, the Child Guidance Clinic, and the Ellen Douglass School for physically and mentally handicapped children.

The history of the Children's Hospital goes back to the year 1909, when a group of dedicated women purchased a house in the north end of the city to serve as a hospital for sick infants. By the year 1912, a 90-bed modern hospital was opened in the north end of the city beside the Red River as the first and only children's hospital in Canada west of Toronto. By 1930, overcrowding and increasing need for space for laboratory, teaching, and research initiated planning for a new children's hospital.

The new Children's Hospital was relocated in the Medical Center and opened in late 1956, with accommodations for 220 beds. In June of 1957, the hospital was officially dedicated at a combined meeting of the Canadian and North Western Pediatric Societies. The present hospital is now in the throes of an expansion program designed to enlarge its facilities by 1967 to 300 beds, with greatly increased space for laboratory or clinical investigation, research, and teaching.

TEACHING ORGANIZATION

The University of Manitoba has the distinction of having had the first full-time chairman of pediatrics in Canada. This occurred in 1950 when Dr. H. Bruce Chown assumed the chair.

There are now 12 full-time pediatric department heads in Canadian medical schools. The head of the department of pediatrics also occupies the position of pediatrician-in-chief to the Children's Hospital and the departmental offices are located in the Children's Hospital. In addition, there is a full-time surgeon-in-chief, who is professor and head of the department of surgery in the School of Medicine. Increasing support from the university and the development of the provincial hospital insurance plan, as well as support from the Children's Hospital voluntary women's guilds, have made possible the creation of full-time teaching, research, and patient care posts in hematology, cardiology, genetics, biochemistry, outpatient department, child development, psychiatry, metabolism, neurology, and immunology. Also, there is a full-time director of clinical investigation and research. These positions carry professorial rank in the pediatrics department.



Fig. 1. First Children's Hospital, 1909 to 1912



Fig. 2. Second hospital, 1912 to 1956



Fig. 3. Present Children's Hospital

PATIENT CARE SERVICES

The Children's Hospital is an example of an "open hospital." This implies that admission privileges are available to any qualified member of the medical profession who applies. The medical staff is divided into an active and courtesy staff. The active staff comprises all the practicing pediatricians in the metropolitan area, as well as physicians qualified in the subspecialties. The courtesy staff comprises all general practitioners on the staff. In fact, only about 10% of medical admissions are general practitioner patients. The medical administration of an open hospital presents many problems, but none are insurmountable. Careful supervision by full-time medical personnel and senior resident staff, clear delineation of treatment privileges within the hospital, and restriction to specialists of care of low-weight infants, meningitis, metabolic disease, and specialized surgery maintain a high level of patient care. Consultations from courtesy to active staff are frequently asked and given. All medical patients in the hospital are available for undergraduate teaching. The residents are encouraged to take responsibility for the care of enough patients in the hospital to ensure satisfactory clinical training experiences. There is an average of 40 public ward or charity medical patients at one time. To these may be added almost an equal number of private ward medical patients for whom residents have responsibility under supervision of the active staff. Responsibility is graded according to experience and demonstrated ability. There is no geographic delineation of so-called "teaching patients."

There are some real advantages in an open Children's Hospital. Among these must be included the frequent opportunities for general practitioners to "rub shoulders" with pediatricians and trained house staff residents. Members of the courtesy staff are encouraged to attend medical staff rounds, grand rounds, and special refresher and postgraduate courses which are held regularly. It is our impression, perhaps difficult to prove, that the standard of child care in the community is better as a result of this type of relationship. One other important benefit should be mentioned. This is the relative freedom in the Winnipeg medical community from

the sort of bilateral paranoia, so prevalent in many areas, which excludes the practicing pediatricians, to all intents and purposes, from the academic circle. Practicing pediatricians in the Winnipeg community are actively involved both in undergraduate and postgraduate teaching programs in a useful and dignified way, although the planning and direction of teaching are the responsibility of the full-time academic staff. As an example, four practicing pediatricians are responsible for a conference with each group of fourth-year medical students on the subject of "child rearing practices." This takes the form of a question-and-answer session following a short introduction by the instructor. Since many of the medical students are now married and some have children of their own, this session is usually a practical, realistic learning experience for all.

A few pediatricians are retained on a part-time basis throughout the teaching year to give regular teaching conferences. These may add up to one or two half days a week and for this the university provides some financial support.

Although the department of clinical investigation and research was officially launched in 1959, members of the medical staff have been carrying on intensive research for many years. Bruce Chown and Marion Lewis began their intensive studies into blood group incompatibilities and hemolytic disease in the infant in the late 1940s and have made fundamental contributions in this field. Along with the Bowman brothers (Wm. D. and John M.) and David Grewar, they formed a centralized diagnostic treatment and research Rh laboratory which, in 1954, with governmental support, undertook the direction of a unique program for hemolytic disease of infancy. Every pregnant woman in Manitoba has prenatal screening of blood specimens early in pregnancy for Rh factor and for red cell antibodies. All exchange transfusions in greater Winnipeg are taken care of by this team in the three larger hospitals, with referred cases being treated at the Children's Hospital. In recent months this group, continuing its cooperation with the department of obstetrics, has set up an active program to study and treat the intrauterine infant who might require intraperitoneal transfusion to reach viability. At the same time, Dr. Alvin Zipursky and Dr. Bruce Chown are pursuing studies using anti-D gamma globulin designed to prevent development of maternal antibodies and thus to prevent hemolytic disease in the newborn due to this cause. If this work leads to the hoped for results, exchange transfusion and even intrauterine transfusion may very well be relegated to medical history.

A comprehensive perinatal mortality study was initiated in 1954 in conjunction with the departments of pathology and obstetrics of the university. This study has recently completed its tenth year. The objectives of this study were threefold: [1] an accurate diagnosis of the cause of perinatal death in every case possible, [2] an assessment of preventability involving the identification of high-risk pregnant women, and [3] the development of an educational program in the community to ensure that available knowledge was being used by all practitioners in the interests of mother and infant. With the development of the department of clinical investigation and research, first under the leadership of Dr. Sydney Israels and more recently under Dr. Alvin Zipursky, research was undertaken in basic and clinical genetics (Dr. Uchida), respiratory failure in infants and children (Dr. V. Chernick and Dr. Adhikari), the application of cardiopulmonary laboratory tests to the physical education program in schools (Dr. Cumming), and the metabolic problems of the neonate (Dr. Haworth). Dr. Haworth, having spent some time in Europe with Prof. Dent in London and Prof. Prader in Zurich, is in the process of developing a program whereby chemical screening of all newborns will detect metabolic errors at the earliest possible time. A two-year study on the effects of maternal cigarette smoking on the offspring has been initiated this year.

Paralleling the upsurge of research activity, the addition of the above full-time subspecialists made possible the development of a more complete residency training program. This involves [1] a three- to four-year residency program leading to certification by or fellowship in the Royal College of Physicians and Surgeons of Canada, [2] pediatric training for general practitioners who wish to improve their skills as family physicians (one- or two-year residencies are offered to such candidates), and [3] training for foreign graduates in pediatrics, either to enter the research field or to return to their own countries with applicable pediatric qualifications.

CHILDREN'S HOSPITAL AND THE COMMUNITY

The Children's Hospital and its medical staff for many years have felt that it should be a focal point for everything that had to do with child health in the province. In addition to the program for the Rh negative mother and infant referred to above, staff members of the Children's Hospital and the department of pediatrics have been involved in: [1] work with handicapped children, [2] a preschool development clinic, [3] a cystic fibrosis diagnostic and treatment center,

[4] a hemophilia consultation clinic, [5] a clinic for study of coagulation disorders, genetic counseling, and genetic surveys, [6] the prevention of rheumatic fever, [7] the education of diabetic children and supervision of the annual diabetic camp, and [8] the direction of a screening program for prevention of mental retardation involving the examination of heel-prick blood of all infants born in Manitoba.

The consultant for the school medical service of greater Winnipeg, the pediatrician to the Children's Aid Society of Winnipeg, the medical director of the St. Amant ward for retarded infants at St. Boniface Sanatorium, and the pediatric consultant to the Society for Crippled Children and Adults of Manitoba are all members of the active medical staff of Children's Hospital and the department of pediatrics. The director of every newborn nursery in greater Winnipeg is a member of the active staff of the Children's Hospital and the department of pediatrics. This has led not only to close cooperation with civic and provincial health services but also has opened facilities for undergraduate and postgraduate teaching important to a comprehensive approach to a study of child health.

THE FUTURE

Pediatrics in general and pediatric teaching in the department of pediatrics at the University of Manitoba in particular are both likely to change their direction somewhat in the next few years. This does not necessarily imply a change in goals. The physician who chooses pediatrics as a career is interested in child health and in children, and he is likely to continue to regard this as his main professional goal. The nature of the residency training program in Canada and the requirements for certification and fellowship make it seem likely that the future trainee in this field will plan to practice essentially consultation pediatrics and leave some or all of the responsibility for well-baby care and management of the common childhood diseases to better-trained general practitioners. Yet we hope there will always be medical graduates who consider total child care a more satisfying medical career.

It seems certain now that by 1967 universal medical care insurance under government auspices will be an accepted fact in Canada. This theoretically means the disappearance of charity patients. It also means that the teaching program of every medical school will have to be reviewed to make sure that an adequate supply of physicians to care for the sick will be made available in Canada and that patients in university hospitals and affiliated teaching hospitals of medical schools will not be hampered by the socioeconomic changes in medical care. The report of the Hall Commission (Royal Commission on Health Services, vols. 1 and 2, 1964), and especially the MacFarlane Report of 1964 (Royal Commission on Health Services, Medical Education in Canada, J. A. MacFarlane, vol. 1, 1964), will inevitably mean large federal expenditures funneled through provincial governments to universities and through universities to medical faculties and affiliated teaching hospitals. The MacFarlane Report states that a teaching hospital costs twice as much to build and at least twice as much to maintain as does a service hospital. It recommends that federal funds be made available to the extent of 50% for the construction and maintenance of teaching hospitals. If these large sums are made available, there will certainly be a surge of new construction of university hospitals and expansion of existing university-affiliated teaching hospitals. The involvement of the university in the financial program of affiliated teaching hospitals will inevitably lead to increased responsibility on the part of the university for the standard of teaching in these hospitals and to a much closer relationship between university and the teaching hospitals administrators. Undoubtedly this will work to the advantage of the medical student and the graduate trainee. At the same time, it will become important to see to it that the standard of community pediatric care is maintained. In our view, this will be possible only if the practicing pediatrician continues to be involved in the teaching program and if opportunities are created for exposure of the general practitioner to continuing education in the field of child care.

Facilities for teaching and research are likely to be much better supported and it should become possible, finally, to slow down the stream of promising young medical graduates who have to seek their careers outside of Canada because of the unavailablity of challenging well-supported posts for them when their training is completed. The outlook for pediatric teaching and research in Canada appears to be brighter than ever, but several political hurdles are still to be overcome, and cooperation of the practicing pediatrician will be necessary as never before.

The Story of the Winnipeg Rh Laboratory

BRUCE CHOWN, M.D.

Winnipeg

THE RH LABORATORY was born of ignorance and brought up in poverty. When Philip Levine propounded his hypothesis of the etiology of erythroblastosis, the disease was unknown to the pediatricians of Winnipeg: in the ten preceding years it was entered only once in the disease index of the Children's Hospital; between the pediatrician and the newborn baby in the maternity wards stood a wall which the then Professor of Pediatrics—Gordon Chown—had cracked but had not yet breached. Once Rh was known, it was manifest that someone in the hospital had to know something about it, had to be able to distinguish between positive and negative, somehow see the babies of Rh-negative mothers and learn about the disease.

It so happened at the time that I was, among other things, Pathologist to the Children's Hospital, so it was incumbent on me to learn about the serological diagnostic methods. In the spring or summer of 1942 by chance I visited Boston and there met Dr. Louis K. Diamond. A few years earlier Lou, with Blackfan and Baty, had written an article which I had much admired: in it erythroblastosis in its various guises had been shown up for what it was, a single villain. In 1942 Lou was busy, both for his own fruitful investigations and for the American Armed Forces, gathering sera of mothers of babies with the disease. At that time there was much confusion about Rh antibodies. They were known as 85%, 87%, 70%, and 30% (today these are anti-D, anti-D+C, anti-C, and anti-E) and Lou offered, if I would send him serum from any woman who had a baby with erythroblastosis, to check it for antibodies and provide me with a good 85% (anti-D) test serum. He was as good as his word, and that fall we had in the Children's Hospital our first 5 ml. of saline-reacting anti-D. We were in business. Or almost.

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The Canadian National Research Council made us a grant of $1,200 "to study erythroblastosis." That was our budget for the year. It went to pay the salary of a young woman, newly trained by two exacting teachers, Professors Dan Nicholson and Alex Cameron, as a laboratory technician, Marion Lewis. Today she is the Canadian authority on the blood groups, but that was November 1942 and we were both beginners. The Hospital gave us a 10 × 10 room in the basement, and now we really were in business.

You get your first customers by advertising. I spoke about Rh at the luncheon meetings of the various hospital staffs, explaining about positive and negative and who was which and what happened next, and offered to do free Rh testing of any pregnant woman in Winnipeg if, in return, I might see the babies of those who were Rh negative. Pretty soon the bloods were coming in and we were going out. For we decided, Marion and I, that if we were to learn anything we had to see the babies fresh and wet and then follow the Rh-positive ones and see what happened to them.

Most times, happily both for the babies and for us, nothing happened to them, for most of the mothers had no Rh antibodies. But we couldn't always be sure of this in advance. Antibody detection methods were still primitive and brought to light only the so-called saline or complete antibodies. It was not until 1945 that methods for the detection of the really damaging, placenta-crossing antibodies became available. And so the only thing to do was to attend the deliveries of *all* the Rh-negative women. This we did, or at any rate we tried to.

Early on I made what turned out to be a lucky mistake: through misreading the directions Lou Diamond had sent me with his anti-Rh serum, we developed the capillary method of Rh typing and antibody detection. Marion brought this to perfection and, in her hands, it proved capable of detecting weak saline antibodies (which often

aeeompanied the then nondemonstrable incomplete antibodies) that could not be detected by the test-tube methods then in use. The method resulted in two things: first, we were less often in ignoranee about the presenee of Rh antibodies in the blood of pregnant women; seeond, it earned an invitation to a meeting on Rh organized in 1944 by Joe Hill of Dallas. At that meeting, the possibility of treating babies with erythroblastosis by means of "exsanguination transfusion" was in the air: Harry Wallerstein, who was to report the first sueh transfusions earried to eompletion, was there, and so was Lou Diamond, who was to introduee the umbilical vein approaeh. Through the generous friendships then established we kept in touch with the developments of this method of treatment. The first result was that the late Dr. Norman Book did, on one of his patients, Romeo Ducharme, the first replacement transfusion in Manitoba on July 24, 1945. The transfusion was a sueeess. Mrs. Dueharme had lost four babies from erythroblastosis.

What a ehange! From being helpless onlookers at the bedside of a baby advaneing to inexorable death, we suddenly beeame aetive partieipants in the baby's defense. Replaeement transfusion was added to the Rh Laboratory's serviee.

Up to this time the serviee had been eonfined to the eity of Winnipeg. In 1948 the Honorable Ivan Schultz, Minister of Health of the Provinee of Manitoba, asked us to extend our serviee to the whole Provincc. This we agreed to do. From that year to the present, the costs, whieh up to then had been met by the National Researeh Couneil and by the St. Agnes Guild of the Children's Hospital, have been borne by the Department of Health. The Laboratory has, however, remained outside the Department, affiliated with the University.

And now followed some still more stirring years. From 1942 to 1947 Marion and I had been present at the delivery of nearly every woman that we knew of that had Rh antibodies. From 1945 on, where we judged replaeement transfusion to be neeessary we gave it. When we extended the serviee to the provinee, Dr. Anna Bryee joined us as a Fellow for a year. We were all young in heart and filled with enthusiasm, and sometimes foolhardiness. In the summer, the flying trips to the eountry were jolly affairs. But winters in Manitoba ean be rough. Sometimes we traveled roads with the snow piled higher than the ear top. Sometimes we stuck in snow drifts. We often reeall one mad venture. Marion and I were ealled one evening about 10:00: A sensitized woman was in labor in a village about 50 miles away. Would we come? It was cold—10 or 20° below zero—but otherwise seemed pleasant enough in the eity. We called in a donor, bled him, and set out. Out of the shelter of the city a driving wind was drifting the highway. A tire went flat. We ehanged it. At the turnoff to the village, the woman's husband was waiting in his truck to break trail for us to the four-bed hospital. Soon the baby was born. We transfused it and lay down to wait for morning. When morning eame, the road we had come by was elosed, and to reaeh home we had to travel south through North Dakota and Minnesota to reaeh an opened highway north. We got pineh ed for speeding just after we reaehed the eity. It was a good life. We eame to have a great regard for the rural general praetitioners of Manitoba; they and their suceessors have remained our friends and eollaborators.

Those days are gone. With the end of the war and the rapid build-up of highly competent residents the treatment of the babies passed into their hands—Harry Bain, Harold Krivel, Ben Shuman, Maurice Berger, the Bowmans, John M. and Wm. D., Dave Grewar—I forget who all. Marion and I did our last replaeement on Mareh 2, 1950.

By then the Laboratory and its expanding staff had moved, first to the deep, dank basement under the Internes' Quarters, elose by the old Children's Hospital, and then to the upper air and sunlight in the Women's Pavilion of the Winnipeg General Hospital. That Internes' basement we had at various times shared with miee—both caged and free-wandering—with rats, pigs, turkeys, hens—*and* with Bill Taylor. Bill, who is now a sober Associate Professor of Pediatrics at the University of Alberta, was wont, when eertain Seottish feast days eame around, to brew a strange and potent brew in the lab and invite eonvivial friends to share both brew and spaee. And then there were the plumbing and the eats, internes' showers overflowing through the eeiling, dog-chased eats, leaping, spitting through the low windows. Life seems so quiet now.

The Rh Laboratory is a monopoly in restraint of trade. We like it that way and so do the does. When we suggested in 1956 that the lab divest itself of the funetions of the assessment and, where treatment was neeessary, of the treatment of the babies, they said "no." And so it was that there were added to the organization that year three part-time "Transfusion Offieers"— the two Bowmans and Dave Grewar—to be responsible for all treatment; they are paid a modest retainer by the Department of National Health. In 1961, as the Laboratory expanded, John M. beeame

Director of Clinical services and Marion Lewis, Director of Laboratory services.

The fields of the Laboratory have expanded in three directions: or perhaps in four: amniotic fluid examination in the assessment of the degree of disease of the fetus; intrauterine transfusion of the fetus threatened by death; the prevention of primary Rh sensitization of Rh-negative women; genetics. John M. led the field in the first and then, from his experience in this field, went on to the second with Rhiney Friesen and then with Cam McInnis and Phil Barnes. We're not orthodox: we have now three obstetricians in the Department of Pediatries looking after the threatened fetuses. And, like the transfusion-officer-pediatricians who are paid a retainer by the Department of National Health for transfusing babies after they are born, these obstetricians are retained by the Department to transfuse fetuses before they are born. Just one big happy family.

Work on the third field—the prevention of primary Rh sensitization—began in 1957 with the support of the National Institutes of Health of the U.S. Public Health Service. For the first three years the going was heavy and not much land broken. That year Alvin Zipursky ("Zip." No relation to Code) yoked the Department of Clinical Investigation of the Children's Hospital up with us and things began to move.

The prevention of hemolytic disease begins to loom as a practical possibility. Another ten years and we'll be out of business.

But genetics will last: until the bomb bursts people will continue to have genes. After that who knows? From traveling the summer and winter roads of Manitoba, Marion and I turned to Indian trails of summer and the Aretic tundra of winter, from heated cars to dog sleds and Bombardiers. And that was fun, too.

At last we have settled down. Here at our doorstep is a genetic field filled with wonder. The simple wild flower is more to be sought after than the exotic.

Neonatal Hypoglycemia: A Six-Year Experience

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Our knowledge of neonatal hypoglycemia dates from 1929 when Van Creveld[1] reported that the blood sugar levels of premature infants were unusually low by adult standards. In 1937 Hartmann and Jaudon[2] suggested that neurological symptoms such as muscular tremors and convulsions in newborn infants might be related to hypoglycemia. From many studies that followed it became established that at birth the normal full-term infant's blood sugar level is approximately equal to that of the mother. During the first few hours after birth the concentration decreases, remains low for several days, and returns to normal adult levels at about the tenth day of age. In the premature infant and the infant of the diabetic mother, the decrease in the blood sugar level is greater and more prolonged. Despite the report by Hartmann and Jaudon, it remained uncertain whether the hypoglycemia was deleterious to the infant until 1959, when Cornblath and associates[3] described 8 infants in whom apnea, cyanosis, coma, and convulsions on the second or third day of life were clearly related to profound hypoglycemia. Reports of similar cases appeared in the literature over the next few years.[4–9]

Symptomatic neonatal hypoglycemia is now generally accepted as an entity of some importance. It is seen in newborn infants who are premature by weight (2,500 gm. or less), but characteristically the birth weight is low for the period of gestation. The smaller of twins is frequently affected[9] and there is a preponderance of males. The infants appear normal at birth but within a few hours or days have tremors, irritability, cyanosis, apnea, or convulsions. Blood glucose levels are found to be less than 20 mg. per 100 ml. and are frequently below 10 mg. per 100 ml. However, some newborn infants who have equally low blood glucose levels do not exhibit these symptoms.

The following is a report of our experience with neonatal hypoglycemia, 1960 through 1965.

PRESENT SERIES

Forty-six newborn infants with hypoglycemia were seen at the Women's Pavilion of the Winnipeg General Hospital and the Winnipeg Children's Hospital during the six-year period (Table 1). The criterion for diagnosis was one or more blood sugar values of 20 mg. per 100 ml. or less. (Blood sugar concentrations were estimated by the "true" sugar method of Nelson and Somogyi,[10,11] by a close modification of it, or by the glucose-oxidase method of Huggett and Nixon.[12]) The patients have been divided into symptomatic and asymptomatic groups, 23 cases in each.

Symptomatic group. These infants all had symptoms associated with their hypoglycemia. In 14 of them it was felt likely that the hypoglycemia was the direct cause of the symptoms because the latter were fairly rapidly relieved by the administration of glucose. This group has been called "idiopathic," which is not an ideal term because there are several clues as to possible etiology. In 3 infants the symptoms could be less definitely related to the hypoglycemia, and it was felt that a central nervous system lesion was probably the primary abnormality. Two of these infants showed little symptomatic improvement with parenteral glucose administration. In 1, convulsions continued and soon showed the characteristics of infantile spasms; the other had a large cephalhematoma and an abnormal electroencephalogram tracing. The third of these infants, who was one of our earliest

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TABLE 1

SUMMARY OF 46 CASES OF NEONATAL HYPOGLYCEMIA SEEN AT THE WINNIPEG CHILDREN'S AND GENERAL HOSPITALS 1960 THROUGH 1965

	No. cases	
Symptomatic		
"Idiopathic"	14	
Secondary to cerebral disease	3	
Infants of diabetic mothers	5	
Liver glycogen disease	1	
		23
Asymptomatic		
"Idiopathic"	22	
Infant of diabetic mother	1	
		23
Total		46

cases, did not receive glucose intravenously but remained lethargic and hypotonic for about two weeks after the blood glucose level had reached normal levels. Five were infants of diabetic mothers and 1 infant had liver glycogen disease. The latter has been reported previously.[13]

Asymptomatic group. Two of these infants were seen recently and blood glucose levels were measured because they were patients who were thought likely to develop hypoglycemia. One was an infant of a diabetic mother; the other showed severe intrauterine growth retardation and is classified as idiopathic. These infants were treated with additional oral glucose administration. The other infants were part of a group of about 80 infants, mostly premature by weight, on whom fasting blood glucose estimations were performed for other purposes. These patients received no special treatment for their hypoglycemia. None of these 23 infants showed any symptoms attributable to hypoglycemia.

CLINICAL AND LABORATORY FINDINGS (TABLE 2)

Sex difference. There were more males in the idiopathic symptomatic group, but there were no sex differences in the other groups.

Birth weight. When plotted on the intrauterine weight chart of Lubchenko and co-workers,[14] many of the infants in the idiopathic groups had low birth weights for their gestational ages (Fig. 1). In 11 of the 14 idiopathic symptomatic cases the birth weights were less than the twenty-fifth percentile for their gestation, and in 6 the birth weights were equal to or less than the tenth percentile. Fifteen of the 22 idiopathic asymptomatic group had birth weights less than the twenty-fifth percentile and 10 were less than the tenth percentile.

Crown-heel length. The crown-heel lengths of 2 infants in the idiopathic symptomatic group were more than one standard deviation below and 1 was greater than one standard deviation above the mean for their gestational ages. The lengths of all the asymptomatic infants of more than thirty-six weeks gestation were below the mean, and the lengths of 6 of them were one or more standard deviations below the mean (Fig. 2).[15] The lengths of the cerebral group and of the infants of diabetic mothers were above the mean for their gestational ages.

Head circumference. The head circumferences of 4 of the symptomatic group and 8 of the asymptomatic group were one or more standard deviations below the means for their gestational ages.[16] The birth weights of 9 of these 12 infants were at or below the tenth percentile.

Of the 16 infants with birth weights equal to or less than the tenth percentile, 7 were one or more standard deviations below the mean in length and 9 had head circumferences one or more standard deviations below the mean. In other words, in approximately half of the low birth weight babies, length and head circumference were proportional more to weight than to gestational age.

Symptoms. In most cases symptoms developed between 24 and 48 hours of age, although in 2 patients the symptoms dated from near the time of birth. The chief symptoms exhibited by the 23 symptomatic infants are shown in Table 3. One infant of a diabetic mother died at 7 days

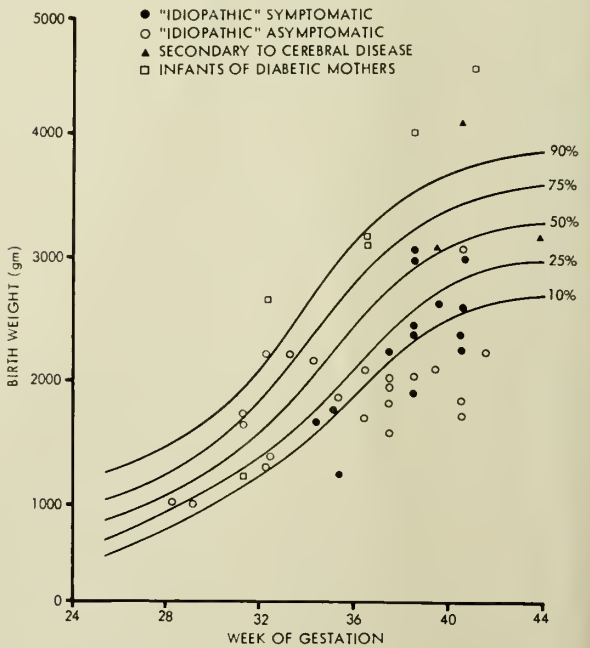


Fig. 1. Birth weight of the hypoglycemic infants plotted against gestational age on the intrauterine weight chart of Lubchenko and co-workers[14]

TABLE 2

CLINICAL DETAILS AND LABORATORY FINDINGS IN 45 CASES OF NEONATAL HYPOGLYCEMIA

Group	No. cases	Sex	Pregnancy	Birth weight Mean and range (kilograms)	Gestation Mean and range (weeks)	Age at onset of symptoms Mean and range (hours)	Hemoglobin Mean and range (gm. per 100 ml.)	Serum calcium Mean and range (mEq. per liter)
Symptomatic								
"Idiopathic"	14	12 M	8 normal					
		2 F	4 toxemia	2.37	38	43	21.7	4.6
			1 APH*	1.26 to 3.08	34 to 40	birth to 72	17.1 to 25.6	3.5 to 6.8
			1 rubella					
Secondary to cerebral disease	3	1 M	2 normal	3.53	41	28	21.6	4
		2 F	1 toxemia	3.15 to 4.15	39 to 43	12 to 48	18.0 to 25.3	3.9 to 4.1
Infants of diabetic mothers	5	2 M	4 normal	3	35	34	17	4.1
		3 F	1 toxemia	1.27 to 4.54	31 to 40	"few" to 48	11.8 to 20.5	2.5 to 6.7
			14 normal					
Asymptomatic			4 toxemia	1.89	35			
"Idiopathic"	22	11 M	2 APH*	1.08 to 3.05	28 to 41	——	——	——
		11 F	2 essential hypertension					
Infant of diabetic mother	1	1 M	1 normal	4.08	37	——	22.6	4.3

*Antepartum hemorrhage

of age and at autopsy the characteristic pathological features were observed. In the others the condition appeared to be self-limited and, in the idiopathic group, symptoms were not seen after 7 days of age. One patient, after remaining well four years, had recurrence of hypoglycemia.

Two infants in the idiopathic symptomatic group had exchange transfusions, one for hemolytic disease of the newborn due to Rh isoimmunization and the other for hyperbilirubinemia of unknown cause. A few of the infants exhibited polycythemia and hypocalcemia (Table 2).

Treatment. During the first year or two the treatment of symptomatic hypoglycemia was not standardized. It soon became apparent, however, that treatment by the administration of glucose by mouth was ineffective in elevating the blood glucose level and controlling symptoms. We also found that sometimes 5% or even 10% glucose given by intravenous drip did not control the symptoms, and we have recorded blood glucose levels of less than 10 mg. per 100 ml. while an intravenous infusion was running. Our present therapeutic regime is similar to that described by Cornblath and Reisner.[17] An initial injection of 50% glucose (2 to 3 ml. per kilogram of body weight) is followed by 15 to 20% glucose by intravenous drip (65 to 75 ml. per kilogram of body weight per day) for forty-eight hours. If further treatment is necessary, we continue with 10% glucose in one-fourth strength normal saline. If symptoms persist after twenty-four hours, hydrocortisone (5 mg. twice daily) and ACTH (4 units twice daily) are given. It is important to taper the intravenous glucose administration because reactive hypoglycemia may occur if it is suddenly discontinued.

Fig. 2. Crown-heel lengths of the hypoglycemic infants plotted against gestational age. (Graph redrawn from data of Gruenwald and Minh[9,15])

TABLE 3

SYMPTOMS EXHIBITED BY 22 CASES OF SYMPTOMATIC NEONATAL HYPOGLYCEMIA

Symptom	*No. cases*
Convulsions	15
Cyanosis	12
Tremors	9
Apneic spells	7
Poor feeding	2
Irritability	1
Muscular hypotonia	1
Dyspnea	1

In the present series, 17 of the symptomatic patients were treated with parenteral glucose and 4 were given corticosteroids in addition. Calcium gluconate was given to those infants with low serum calcium levels.

Prognosis. Thirty-two infants have been followed up at ages varying from 23 weeks to 4 years (Table 4). The methods used for the follow-up examination have previously been described.[18] This study is continuing and the results can only be considered as preliminary, since many of the patients are still too young for any final opinion to be given. However, of the 17 infants in the symptomatic group who have been followed up so far, 6 are mentally retarded and show unequivocal abnormal central nervous system (C.N.S.) signs and another, although showing normal development, has abnormal C.N.S. signs. Three show possible mental retardation, but the children are still too young for final assessment. One child, as already mentioned, was apparently normal in every way until the age of 4 years, when he was readmitted to hospital in hypoglycemic coma. He rapidly recovered with treatment, and carbohydrate function tests showed no abnormality. Seven of the 17 symptomatic hypoglycemic patients appeared to be normal on follow-up examination. Thirteen of the 15 asymptomatic cases followed appear normal; 2 show possible retardation.

DISCUSSION

In general, our experience with neonatal hypoglycemia is similar to that of others. While it must be recognized that hypoglycemia may be secondary to primary neurological abnormalities, liver glycogen disease, galactosemia, fructose intolerance, maple syrup urine disease, and pancreatic islet-cell tumor, the majority of cases are not due to these conditions. Hypoglycemia in the infant of the diabetic mother has now been clearly shown to be due to hyperinsulinism. The islets of Langerhans of these infants are hypertrophied and plasma insulin levels are elevated.[19,20] When hypoglycemia is not due to these other disorders, it is seen characteristically in the malnourished or growth-retarded infant whose birth weight is low for the gestational age. In many cases, the cause of the intrauterine growth retardation is unknown, but toxemia of pregnancy, abnormalities of the placenta, and smoking during pregnancy are sometimes etiological factors. They may have polycythemia, and serum calcium levels may be low. Although the etiology of the hypoglycemia in this group of infants is not by any means understood, there are a number of possible etiological factors.

Liver glycogen stores increase greatly during the latter third of pregnancy, and at birth the full-term infant has approximately twice the average adult content of glycogen in the liver. Most of the glycogen is lost during the first few hours following birth, as the infant uses it to supply energy during the time he will be receiving little or no food.[21,22] The premature infant is born with lower glycogen stores because the pregnancy has been shorter and there has been less time to accumulate the stores. There is also evidence that the growth-retarded infant has low carbohydrate stores in the liver and other tissues.[7] One of the infants in the present series was given an injection of epinephrine when hypoglycemic and another was given glucagon. In both of them only a minimal rise in blood glucose level was observed, indicating impaired

TABLE 4

FOLLOW-UP DATA OF 32 CASES OF NEONATAL HYPOGLYCEMIA

Group	*No. cases followed*	*Outcome*
Symptomatic		
"Idiopathic"	12	3 abnormal C.N.S. findings and retardation
		2 possible retardation
		1 recurrence of hypoglycemia at 4 years; normal C.N.S. and development
		6 normal
Secondary to cerebral disease	2	1 abnormal C.N.S. findings; normal development
		1 infantile spasms and retarded
Infants of diabetic mothers	3	2 abnormal C.N.S. findings and retarded
		1 normal
Asymptomatic	15	2 possible retardation
		13 normal

glycogenolysis. These babies with glucose therapy made rapid recoveries from their hypoglycemia; it seems likely the failure to release glucose from the liver was due to lack of glycogen.

It has been pointed out that, whereas the full-term infant at birth has a brain that is 3 times heavier than the liver, in the malnourished infant the brain may be 7 or 8 times the weight of the liver.[23] It has been suggested that in these infants the metabolic needs of the relatively large brain may exceed the capacity of the liver to produce glucose.[24] However, in this series, more than half of the infants whose birth weights were in the tenth percentile or less for their gestational ages had head circumferences of one or more standard deviations below the mean, suggesting that their brains were not proportionately large. Little is known about insulin release in these infants. There may be an exaggerated insulin release mechanism which is in contradistinction to normal full-term infants who show a striking pancreatic islet unresponsiveness to a glucose load.[7,19,20]

It is not understood why some hypoglycemic infants should develop neurological symptoms while others, with equally low blood glucose levels, should be asymptomatic. It may depend upon the length of time during which the infant is hypoglycemic, as Baens and associates[25] have suggested, but we have no information to support this. In many of our asymptomatic group of infants, fasting blood glucose levels of 20 mg. per 100 ml. or less were recorded on 2 or 3 different occasions, but isolated blood glucose estimations obviously cannot reflect the fluctuations that may occur over a twenty-four-hour period. Cornblath's group seldom find hypoglycemia of this degree in asymptomatic premature infants, but his experience is at variance with ours. Yu and co-workers[26] have recently reported that in 6 healthy premature infants weighing less than 1,500 gm. at birth, blood glucose levels of 20 mg. per 100 ml. or less were found in every one at some time during the first forty-eight hours of life. There is some experimental and clinical evidence to suggest that the high blood lactate levels which are commonly seen in newborn infants may protect the brain against the effects of hypoglycemia.[27,28]

The results of follow-up examinations on the infants in the present series, although preliminary, indicate the high incidence of permanent neurological sequelae in symptomatic hypoglycemia. For the past year or two we have recommended blood glucose estimations in any baby in our nursery developing neurological signs, unexplained apnea, cyanosis, and tremulousness, especially in growth-retarded infants. It is of some interest that 5 out of 6 of our cases seen during the past year were normal on follow-up examination and the sixth showed only possible impairment on the first testing. Time may tell whether this suggestively better prognosis in our most recent cases is a reflection of the greater awareness, earlier diagnosis, and more efficient treatment of the disorder in this center. The preliminary results of follow-up examinations in the asymptomatic hypoglycemic infant suggest that the prognosis in this group may be no different from that of prematurity alone.

One of our patients had a recurrence of hypoglycemia at the age of 4 years. We have seen 2 other children, in whom spontaneous hypoglycemia was diagnosed at 14 and 18 months of age, respectively, who had intrauterine growth retardation and symptoms in the neonatal period which could have been due to hypoglycemia. None of these patients had liver glycogen disease or any other recognized cause of hypoglycemia. Neligan[29] has had a similar experience of patients with neonatal hypoglycemia having recurrences in later childhood. These cases can be presumed to be examples of "idiopathic spontaneous hypoglycemia of childhood" presenting in the neonatal period and emphasize the importance of adequately following up cases of neonatal hypoglycemia.

SUMMARY

Forty-six cases of neonatal hypoglycemia seen during a six-year period are reviewed, and the clinical and laboratory findings are reported. Half of these patients had symptoms associated with their hypoglycemia and half were asymptomatic. In 3 infants the hypoglycemia was probably secondary to neurological disease; 1 infant had liver glycogen disease and 6 were infants of diabetic mothers. In 36 infants the hypoglycemia was "idiopathic," and many of these showed intrauterine growth retardation. Thirty-two infants were subsequently followed up and a high incidence of neurological sequelae was found in the symptomatic group. Some of the etiologic factors in neonatal hypoglycemia are considered.

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(Continued on page 61)

Teaching Resuscitation in a Children's Hospital

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THE DEPARTMENT of anesthesia must take the responsibility for teaching and organizing resuscitation in a teaching hospital. This paper is an account of our present practices, evolved over some nine years since the opening of the present hospital. If it should seem too positive and even narrow, this may be because it is based on close and realistic experience and on disappointments and failures, as well as successes. Certainly, it does not claim to be the last word.

TEACHING, ORGANIZATION, REVIEW

Our activities have been of three different kinds, namely, education of personnel, organization of equipment, and—absolutely essential—frequent reviews of results. In looking for possible reasons for failure we have found it a great help to have those patients who have received some form of resuscitation marked out on the monthly mortality review of the records department. The records of all these patients are then examined to determine whether any improvement could have been made.

From such a review two facts immediately stand out. First, some attempt at resuscitation is carried out much more often than one might guess at first. Those obviously dead on arrival or those beyond hope of revival when the doctor arrives on the ward are still frequently subjected to part or all of the ritual. One should never discourage this. After all, judgment comes with experience only, and a too flippant attitude would be worse.

The second inescapable fact is that the important part of resuscitation is usually done by relatively unskilled staff, often by nurses and junior interns. If an anesthesiologist can attend, there should be no problem in carrying out the necessary maneuvers at once. Unfortunately, it is the vital first few minutes that count, and the person involved is most likely to be the ward nurse, the nurse in casualty admitting, or the very junior interns. These, then, are our prime targets.

The program we run is open to all, even to our colleagues on the pediatric staff. Since they have to deal occasionally with airway problems on house calls, it is a practical problem for them.

On occasion our confreres have been guilty of sending junior interns to us for "intubation sessions." There are two things wrong with this concept, both of them basic. First, immediate resuscitation does not usually require intubation. Second, no one can be taught to intubate with speed and confidence in two or three sessions or to retain the knack without frequent practice.

PRESENT TEACHING PROGRAM

Nurses—a question of attitude. Formal education on this subject must begin in the student nurses' first year. We have written a manual on the subject for our nurses and junior intern staff. This is required reading.

It is extremely important to make this teaching of student nurses a formal part of their education. They will come to realize right away that they will be expected to perform resuscitation as part of their usual and routine duties and that they will be trained accordingly. This avoids the common attitude toward resuscitation as an unpleasant and unusual occurrence, associated with some degree of panic and even recrimination, and an event to be quickly banished from mind as soon as they get off duty.

Organized nursing, particularly in the United States, is very concerned with possible medicolegal implications. This issue simply cannot be avoided. Professional staff, nurses included, are expected to deal with life-endangering conditions as best they can until medical help arrives. They must be expected to begin pulmonary ventilation by mouth-to-mouth breathing or the Ambu bag and to do external cardiac compres-

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sion. Indeed, this is little more than first-aid workers do. To be so afraid of the law as to confine oneself to telephoning for help will completely arrest progress.

Instruction to nurses. Our manual is made available to all, and we expect it to be studied and discussed. Formal lectures and demonstrations are arranged through the department of nursing education at least twice a year.

We keep "open house" in the operating room for nursing and intern staff. Head nurses on the wards know this and come themselves or send their junior staff when problems occur. They can refresh themselves in this way on any part of the theory and practice of resuscitation. This facility is not used enough, but one sees sporadic outbursts of interest and enthusiasm, spiked with anxiety, following some distressing incident. This makes the effort worthwhile.

Finally, but most important, there is the question of "training the teachers." The nursing staff can be properly educated only through their own schools and in-service program. Particular effort should be concentrated on the school, where it is absolutely essential to get support and enthusiasm.

"Elite" training. The nursing staff in the recovery room and in the intensive care unit needs special training and attention. The rotation of junior staff through these areas offers a special opportunity to drill them thoroughly.

Junior intern staff. The chief resident in pediatrics arranges a program for these doctors as soon as they arrive in hospital. We have a lecture and discussion on the basic principles and procedures and then show a film specially made at this hospital. This is followed on successive mornings by demonstrations and practice in the operating room for groups of two or three at a time. For this we use the models (mouth-to-mouth breathing and external cardiac compression) and the unconscious, paralyzed patient (the Ambu bag and mask).

BEHAVIOR PATTERNS IN CRISES

Repeated observation of the train of events in desperate circumstances has convinced me of a number of things; the first of these is the great need for simplicity. Luckily, effective resuscitation is based on very simple principles.

Everyone associated with medical practice has disciplined himself to resist panic and hysteria. Nevertheless, movements tend to become either frozen or repetitive and purposeless; a trance-like state of inertia can set in. The room becomes cluttered with onlookers. We have a rule that the person in command, whether a nurse or a doctor, has authority to dismiss all but essential helpers. The reactions of those at the front of the throng are often more energetic than intelligent. A great anxiety to help is very naturally and laudably manifest, but the tendency is to go to the telephone, to the wall oxygen and suction, to the drug cupboard, to the resuscitation tray, in fact, away from the patient. The thing to do is to bring the simplest effective measures to bear on the patient with the utmost of speed.

KNOWLEDGE AND PERFORMANCE REQUIREMENTS

There are only three simple steps to immediate resuscitation: [1] clear the airway, [2] ventilate the lungs with oxygen, and [3] restore the circulation.

Clearing the airway. We stress the extreme importance of two simple pieces of equipment: a metal tonsil suction and a foreign body forceps. The Magill so-called intubating forceps is nearly ideal. Plastic or rubber suctions require two hands, are difficult to direct, and are less effective than the metal suction. Even in small infants the metal suction is preferable at this stage from every viewpoint.

Ventilating the lungs. Oxygen for resuscitation comes from three main sources—expired air, room air, and the usual cylinder or piped supply. The importance of the last of these is greatly overestimated. Too much time is lost in connecting up to wall oxygen, to tanks, or to fiddling with flow meters, while room (21%) and even expired air (16%) contain enough oxygen to bring back to life almost all who are within recall. Expired air is used in all forms of mouth-to-mouth breathing, and room air is used with the Ambu bag, which also comes with an attachment for oxygen. Mouth-to-mouth breathing must be learned on the models, adult and infant size. Use of the Ambu bag is learned on the unconscious, paralyzed patient under anesthesia.

Restoring the circulation. The important steps are to decide when the circulation is not adequate, to carry out effective measures, and to know the important signs of success or failure. Absence of a palpable pulse in a major artery such as the carotid means that the circulation is inadequate. The stethoscope and electrocardiograph are unreliable and wasteful of time.

We teach external cardiac compression on the model and point out that it is generally done poorly these days because of an unfortunate fear of complications, direful accounts of which fill the literature. Compressing the chest and heart between two hands, one over the sternum and the other, palm up, behind the vertebral column, gives better control than any other technique we

have used. Certainly, we believe the reports that it is possible to compress the heart between the fingers and thumb of one hand in a small premature infant, but this is too fanciful for practical teaching. The heart lies slightly higher in the newborn because of the high-arced diaphragm; one presses back over the middle third as compared with the lower third in the older child.

Success or failure. If resuscitation is doing some good, there are two signs which should be obvious at once. There is lessening of cyanosis and dilated pupils become smaller. If these do not occur, then more effective measures are needed or the patient has been dead too long.

Some exceptions and refinements. If we reach this level of knowledge and performance with our nurses and junior interns we are well satisfied. However, there are exceptions, refinements, and extensions which are described in some detail in our basic manual and in discussion with those who show further interest. This applies particularly, of course, to the teaching staff of the nursing school. These girls demand more theory and more complete and academic background knowledge. This is excellent, since it makes them better teachers, but they should not try to instill every detail into their pupils.

Mouth-to-mouth breathing will not work where, for example, there is complete laryngeal obstruction from inflammation or a foreign body. A tracheostomy may be needed right away.

External cardiac compression is useless in flail chest or with cardiac tamponade. Open chest cardiac massage is required.

The electrocardiograph is necessary to diagnose causes of failure of the heart to resume sinus rhythm. A small and simple model is kept in the casualty department. External and internal cardiac defibrillators and pacemakers are also stored here and these may be essential on occasion. Nurses must at least know what this equipment looks like, where it is kept, and how it is used.

The place of drugs (a very small one in immediate resuscitation) and the use of general body hypothermia (which must be managed with the utmost care) are discussed with those seeking a more advanced level of knowledge.

Types of emergencies seen. Trauma and infection account for most emergencies. Burning, electrocution, smoke inhalation, drowning, and head injuries from car accidents are often seen in casualty. Patients comatose from ingestion of poison or with an obscure diagnosis may not need emergency resuscitation but they do need intensive care. The stomach is never washed out in an unconscious patient until the airway is protected by endotracheal intubation. Infections leading to obstruction of the airway are particularly treacherous, as are foreign bodies in the larynx or trachea. Nurses are taught that if the patient is getting along, even with difficulty, not to try to dislodge a foreign body by thumping the back. This can turn a partial bronchial obstruction into a complete and fatal laryngeal one.

The use of a teaching film. The motion picture we have made, "Resuscitation in a Children's Hospital," completed in 1964, holds something of a special place. It was entirely produced in this hospital by the departments of photography and anesthesia and involved wide participation by the staff. It scores well as a teaching help in two ways. First, the interest aroused in seeing these events calmly dissected and logically described is usually quite profound. Secondly, the cast of hospital personnel gives the film a local flavor and personal appeal. Motion pictures are of dubious value for retentive teaching, but this one serves the purpose of stimulating and even irritating people into thinking more. Incidentally, it has achieved some national and international notoriety on both sides of the Iron Curtain.

Have our results improved? The honest answer has to be that we do not know. The spectrum of cases seen from year to year varies so much in kind and degree that comparisons are often meaningless. The work is tedious in some ways and rewards are very difficult to assess, but there can be no letting up on the effort.

SUMMARY

The department of anesthesia must take care of resuscitation, teaching personnel, organizing equipment, and reviewing results.

Teaching must be beamed primarily at nurses and junior house staff, since they are usually the first to see patients in emergencies and have the best chance to improve results. Help of the School of Nursing is essential. Education must commence in the student nurse's first year.

We use a specially written manual and film, lectures, discussions, practice, and demonstrations on models and in the operating room.

Resuscitation procedures must be kept simple —clearing the airway by metal suction and foreign body forceps, inflating the lungs by mouth-to-mouth breathing or the Ambu bag (preferably), and external cardiac compression. These are basic requirements, the limitations of which must also be known.

The use of drugs, hypothermia, electrocardiograph, defibrillation, or heart stimulation seldom has any immediate place but must be taught in this perspective to round out knowledge.

Strabismus: A Review of 100 Surgical Cases

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THE MOST COMMON ocular problem of childhood is strabismus. Numerous reviews show that about 3% of all children have this condition at birth or develop it during early childhood. Premature children have a higher incidence of strabismus, and the rate is about 12% in children with cerebral palsy.

It is important that children with strabismus should be seen by an ophthalmologist as soon as the condition is recognized. In the first place, strabismus is occasionally produced by diseases such as retinoblastoma, glioma of the optic nerve, cerebral tumors, or intraocular disease, such as cataract or macular choroiditis. Secondly, in most cases in which the eyes are otherwise normal, the dominant eye is used exclusively and the deviating eye becomes blind from lack of use. The longer the condition is allowed to persist, the more difficult it is to treat. If the child is not seen before the age of 6, the eye usually remains blind or practically so for the remainder of the patient's life. Therefore, early recognition, investigation, and treatment are of paramount importance.

Recently, a study was made of a consecutive series of 100 children with strabismus who attended the public outdoor department of the Children's Hospital and who were treated surgically. The survey did not include those cases of accommodative convergent strabismus due to extreme hyperopia which were cured by the use of spectacles.

RESULTS

Our results are similar to those obtained in other studies, that is, a preponderance of convergent strabismus (Table 1). Generally, divergent strabismus in children is intermittent in character, with one eye diverging only when the child is tired or is looking into the distance.

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There was no sex difference in the case of convergent strabismus but there appeared to be a sex difference in the divergent form. This was probably more apparent than real, and the numbers were not sufficiently large to be significant. Parents tend to be more aware of a daughter's appearance, and it may be that this is the cause of the apparent preponderance of females with divergent strabismus.

The age of onset is often difficult to determine with certainty because parents forget when the squint was first noticed (Table 2). In 21 cases, the parents were unable to say when it first became apparent.

Despite the fact that in 68 cases the strabismus was observed during the first two years of life, 31 patients (Table 3) were not seen until 6 years of age or even later. This neglect is disastrous because, unless occlusion of the dominant eye is started well before the age of 6 and soon after the squint develops, the amblyopia of disuse cannot be effectively overcome.

MANAGEMENT

Investigation. An inquiry should be made into the family history, because the condition is hereditary in at least 50% of the cases. On other rare occasions, there may be a family history of retinoblastoma. This condition may well cause strabismus and should always be considered. In glioma of the optic nerve or a cerebral tumor, particularly an astrocytoma of the cerebellum, strabismus may be the presenting symptom so that the ophthalmologist may be the first doctor to see these children. Intraocular disease also may cause strabismus, and conditions such as congenital cataract, macular choroiditis, and hereditary diseases of the macula must be excluded. These conditions can be recognized by dilating the pupils widely and examining the fundi with an ophthalmoscope.

Refraction. If a refractive error is present, it must be corrected by glasses. In many cases of accommodative convergent strabismus, the wearing of glasses alone will correct the deviation

TABLE 1

DISTRIBUTION OF STRABISMUS

	Males	*Females*	*Total*
Convergent	42	44	86
Divergent	4	10	14
	46	54	100

TABLE 2

AGE AT WHICH STRABISMUS WAS FIRST OBSERVED

	1st year	*2nd year*	*3rd year*	*4th year*	*Unknown*	*Total*
Convergent	42	18	7	3	16	86
Divergent	6	2	1	0	5	14
Total	48	20	8	3	21	100

TABLE 3

AGE OF FIRST ATTENDANCE AT EYE CLINIC

	1st year	*2nd year*	*3rd year*	*4th year*	*5th year*	*Older*	*Total*
Convergent	8	15	19	12	8	24	86
Divergent	0	1	2	2	2	7	14
Total	8	16	21	14	10	31	100

TABLE 4

METHODS OF TREATMENT

	Glasses	*Occlusion*	*1 operation*	*2 operations*	*3 operations*	*4 operations*
Convergent	73	42	55	24	6	1
Divergent	3	0	11	3	0	0

TABLE 5

RESULTS OF TREATMENT

	Cured	*Cosmetically straight*	*Cosmetically poor*
Convergent	13	64	9
Divergent	3	10	1

and, in the majority of other cases, the provision of glasses will reduce the angle of deviation. In our series, glasses were considered to be advisable and were prescribed in 76 cases (Table 4). Glasses must be worn constantly—even after the eyes are straightened by surgery. In fact, it is frequently necessary to wear glasses at least until the late teens when, in some cases, there is a reduction in the degree of hyperopia.

Occlusion or patching. In our series, 42 patients required occlusion (Table 4). By this we mean that the fixing or dominant eye is covered all day every day for one, two, and sometimes three months. In this way, the child is forced to use the lazy eye and the amblyopia of disuse is overcome. Occlusion should be continued until the vision in the two eyes is equal. Surgery is then indicated. If patching is started soon after the strabismus develops, it is surprising how quickly the child regains vision in the squinting eye. The parents are sometimes disturbed to find that the strabismus is reversed, with the previously fixing eye becoming the deviating eye. This indicates that the vision in the squinting eye is normal and that the time has arrived for the eyes to be straightened by surgery.

However, if patching is delayed until after the age of 6, it is usually too late for it to be of any real value. When there is no improvement in vision in the squinting eye after a period of occlusion, then surgery should be done to improve the cosmetic appearance.

Operation. As seen in Table 4, one operation sufficed in 66 cases and 2 were required in 27 cases, but 3 were necessary in 6 cases, and one patient needed 4 operations. Therefore, when discussing the question of surgery with parents, they should be made to understand that the deviation might not be corrected with one operation. Strabismic surgery is unlike an appendectomy. It is possible after an appendectomy to say with certainty that the child can no longer be affected with appendicitis, but a similar guarantee cannot be given that one operation will correct strabismus. The degree of correction of the angle of strabismus given by a 4-mm. recession of a medial rectus varies greatly from one patient to another, depending upon the size of the eye, the length of the eyeball, the strength and elasticity of the muscle, and the toughness of the intermuscular septa. All these factors must be assessed by the surgeon at operation, and it is not surprising that he may be compelled to operate a second or even a third time in order to obtain a good cosmetic result.

Orthoptic training. A decade ago, orthoptic training both before and after operation was widely used in the treatment of strabismus. It is still used today, but much less frequently. The reason for the change is not far to seek. For a generation, ophthalmologists have been emphasizing the need for early diagnosis and treatment of strabismus. Therefore, there is a trend for parents to bring their children to the ophthalmologist at an earlier age. In private practice, children over age 6 with long-established squints are seen much less frequently. Most are now seen at the ages of 2 or 3. Since a child must be age 5 or older for adequate cooperation in orthoptic training, it is obvious that the demand for pre- and postoperative orthoptic training is diminishing. By the time the child is 5 or 6, the squint should be either completely cured or

(Continued on page 63)

Some Observations on Eskimo Health

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OUR TRUE NORTHERNERS—the Eskimos—have earned universal admiration for unique adjustment to their environment over the last seven to ten thousand years. Today, the total Eskimo population is about 60,000 to 70,000. Of these, approximately 30,000 live in Greenland under Danish rule. Another 20,000 or so live in Alaska and are citizens of the United States. Several thousand Eskimos live near the northwestern tip of Siberia. The remaining 13,000 are thinly scattered along the coastline of northern Canada from the Yukon to northern Quebec.

Increasing archaeologieal studies in the Arctic over the past forty years are giving a clearer picture of the origins and migrations of the Eskimo. A surprisingly large amount of material has been published since their first contact with Europeans. This has been masterfully summarized in 3 recent publications by Diamond Jenness.[1–3]

In 1945, the Department of National Health and Welfare assumed responsibility for the health of the Canadian Eskimo. Since that time, it has dotted the Arctic with hospitals, nursing stations, and health stations. In 1961, the Canadian government spent over $3 million (100 times the amount spent in 1939) to provide the Eskimos with medieal care. This amounted to an expenditure of $285.31 for each Eskimo, broken down as follows: hospitalization, $199.29; transportation to the hospital, $24.50; local health services at nursing stations, $57.78; and administration, $3.74.

In 1961, the Eskimo birth rate was 59 per 1,000, compared with the Canadian national average of 26.9 per 1,000. The infant mortality rate was 193 per 1,000 live births, compared with the Canadian national average of 27 per 1,000. Dr. J. S. Willis[4] suggests 4 reasons for this extraordinarily high mortality rate: "[1] They are being exposed as never before to a wide range of infections. [2] Since most of them do not have the benefit of continuous warmth and shelter, of constantly adequate nutrition, and of a sanitary environment, they are extraordinarily vulnerable to these infections. [3] Laid low by these infections, the conditions of their environment aggravate their disease. Since the same infeetions often afflict their parents, they may go from bad to worse beeause of diminished parental eare. [4] Only a few have the benefit of immediate medieal care and many never receive any such care."

After many years' experience in the Arctic, Jenness[2] writes: "It is not too difficult to bring up a baby in a warm house with running water, sanitary facilities, and a nurse or a doctor in the neighborhood, but it is infinitely more difficult to raise it in a tent or a snow hut far removed from any settlement, without heat save from a small primus stove that must cook the food and melt all the snow or ice that is needed for drinking water, while the temperature outside may range anywhere from the freezing point to 50° F. below zero. I have seen a mother eroueh down in the lee of her sled, during a winter migration, withdraw her naked baby from under her fur coat, and calmly change its tiny caribou-fur diaper, although the temperature was 30° F. below zero and a thirty-mile-an-hour gale was whipping the snow against our faces. It seemed miraculous that either the baby or the mother could survive such exposure."

In 1963, the Canadian Pediatric Society set up an ad hoe Indian and Eskimo Child Health Committee under the chairmanship of Dr. John Charters. This committee has worked in collaboration with the Department of National Health and Welfare, with a view to reducing the mortality and improving the health standards of the native children. As a result of their combined efforts, schemes have been worked out in several

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provinces whereby pediatricians from such places as Montreal, Kingston, and Winnipeg tour the Arctic. They serve as advisors in regard to general pediatric health matters and function locally in matters of child health and education. Pediatric resident training is being incorporated into this scheme.

In February 1965, with the help of Dr. Otto Rath, regional director for medical services, Department of National Health and Welfare, and his staff, I was able to visit Eskimo communities in the north central zone of the central region. This is a vast dome-shaped area north of Manitoba populated by about 2,000 Eskimos and several hundred whites (Fig. 1). It includes some of the most inhospitable land on Earth—the eastern portion of the great Barren Grounds and also Southampton Island at the northern extremity of Hudson Bay. Transportation was by scheduled airline from Winnipeg to Churchill and by chartered plane from Churchill northward. The zone has 4 nursing stations, located at Coral Harbour, Baker Lake, Rankin Inlet, and Eskimo Point, and one hospital operated by the Grey nuns at Chesterfield Inlet. The communities of Repulse Bay and Whale Cove have lay dispensers. The nearest physician is in Churchill.

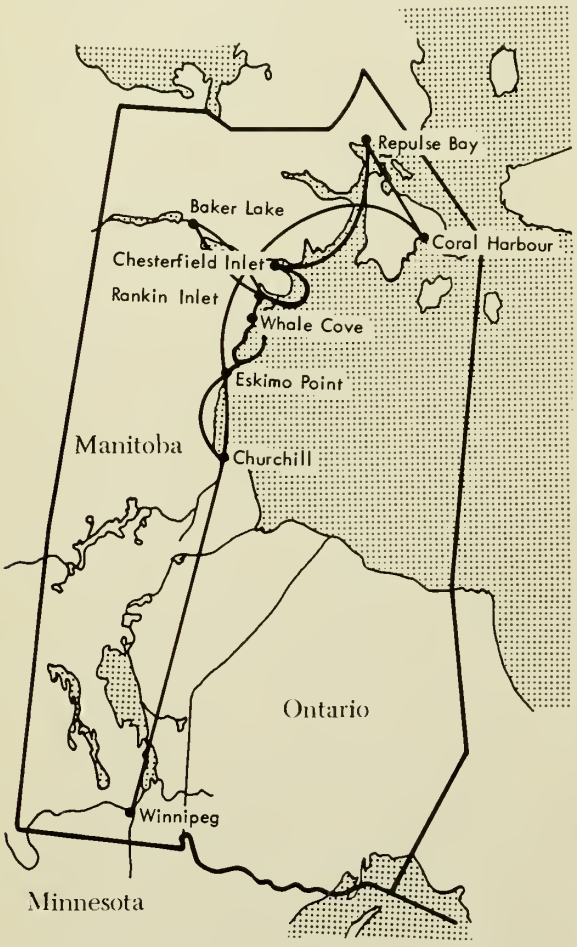


Fig. 1. Map showing tour of the north central zone, which is populated by 2,000 Eskimos and several hundred whites

COMMUNITY FACILITIES

Coral Harbour. Located on the southern shore of Southampton Island, Coral Harbour is some 635 air miles northeast of Churchill. At the time of the tour, it had a population of 256 Eskimos and 23 whites. Twenty men were stationed 8 miles away at the Department of Transport airport. There were 112 Eskimo adults; 18 infants, up to 1 year of age; 66 preschool children, 1 to 6 years of age; 51 schoolchildren; and 9 "outside" at school. Some 5 or 6 of these families lived in Snafu, a small "suburb" 3 miles away, created and named by a neighboring World War II U.S. air force base.

The Eskimos of Southampton Island are largely self-sufficient. They hunt and fish both winter and summer, and most Eskimos had retained their dog teams in preference to the Skidoo, which has replaced dogs in many Arctic communities. Housing consists of one-room prefabricated homes which are subsidized by the government.

The nursing station was well constructed and adequately equipped. It was divided to separate the nurses' living quarters from the treatment areas. There were 2 rooms, each with 2 beds, one of the rooms being designated for maternity, and a large well-arranged work area. This nursing station was staffed, as was every station I visited, by remarkably well-trained and dedicated nurses who were doing a magnificent job, assuming heavy responsibilities, and carrying on in a manner of which their colleagues, and the medical profession generally, may be proud.

All preschool children were given multivitamin supplements as well as supplemental iron drops. These were replaced by vitamin capsules for all the schoolchildren. The diet of the Eskimo (including children over 2 years of age) in order of importance or volume was seal, char, walrus, polar bear, square flipper (a big seal), white whale, and cod. Only the skin (muktuk) of the whale is eaten. Bannock, made from flour and lard purchased at the Hudson Bay Company store, ranks fifth or sixth in the list. Mothers nurse their babies for one to two years, unless they have another one, and then give them powdered milk purchased at the Hudson Bay store.

Monthly immunization clinics are attended by virtually all the children. Routine diphtheria, tetanus, pertussis, poliomyelitis, and smallpox

inoculations were given, and there was a recently inaugurated measles immunization program using live attenuated virus with gamma globulin. To prevent tuberculosis, BCG vaccine was given to all newborns. A similar immunization scheme was in effect at the other settlements visited, with the exception of Repulse Bay and Whale Cove, where no regular immunization was available. In Rankin Inlet, it was lagging at the time of my visit.

Aside from extensive dental caries, the most common medical problems among the children were purulent ears, respiratory infections, and impetiginous rashes. This was more or less true of the entire area visited. The physical and emotional problems of the schoolchildren will not be discussed. It must be remembered that most of the Eskimos spoke virtually no English.

All emergency medical care is provided by the 2 nurses who contact the nearest physician at Churchill by radio, if necessary, when weather permits. Patients are evacuated to Churchill by scheduled bimonthly flights or by emergency charter flights, weather permitting. It was not until I found myself diagnosing and treating a case of bacterial meningitis by radio that I really comprehended the meaning of "weather permitting."

During 1964, there were 2 deaths at Coral Harbour. An older man with massive gastrointestinal bleeding was evacuated to Churchill where he died before surgery, after transfusion of some 20 pints of blood. An 11-month-old child died very quickly at the nursing station of what was probably meningitis.

Repulse Bay. The most isolated community in the zone, the 134 Eskimos and 3 whites have no scheduled transportation arrangement with the South, aside from the yearly supply boat. The Roman Catholic missionary acted as lay dispenser, with occasional advice by radio from Churchill. Emergencies were flown to Churchill when this was possible.

The diet of the Eskimo, in order of volume, was seal, caribou, walrus, char, white whale skin, and flour plus powdered milk for infants when necessary. The blubber of the whale was used for heat and light and the meat, for dog food. Ten government-supplied, one-room houses were erected in the fall of 1964. Ten more were constructed by the Eskimos from the crates in which the prefabricated houses were shipped.

There were two deaths from Repulse Bay in 1964. An old man died of what was diagnosed as cancer in a Manitoba hospital, and a stillborn baby was delivered of a lady who had been evacuated to Churchill.

Chesterfield Inlet. Here there were 144 Eskimos (Fig. 2), plus 54 living at Daly Bay, a fishing community 70 miles away. There were 34 whites. A hostel housed 82 students, mainly from Igloolik, Pelly Bay, and Repulse Bay.

Medical care for the community was provided by the Grey nuns at the Sainte Therese Hospital, which was once the only medical facility for the entire zone and now is an almost empty three-storied building. Vitamin supplements were provided for preschool and schoolchildren. The diet of the Eskimo consisted of food bought at the Hudson Bay store—bannock and some canned meat—supplemented by what little caribou and fish they could get. The majority of the Eskimo were unemployed, surviving on government relief.

There were 3 deaths during 1964—all children. One was a 2-month-old baby with a cleft palate who died of unknown causes after a one-day illness. The second was an 8-month-old baby who probably had pneumonia. The third also was an infant who died under uncertain circumstances at Daly Bay. There were 14 deliveries in 1964, with no stillbirths and no neonatal loss.

Rankin Inlet. This was the site of a nickel mine that had ceased operations some two years before. Of the 287 Eskimos living here, over 50% of the adults were on welfare. Housing was in the standard prefabricated homes at the "Eskimo village." Food, largely bought at the Hudson Bay store, was supplemented moderately by caribou and fish and liberally by beer, also purchased at the store. This was the only settlement in the zone where beer or liquor was sold.

The 6-bed nursing station was located in the bunkhouse of the defunct mine and was inadequately staffed, with only one nurse. Vitamin supplementation was similar to that at Coral Harbour.

Three of the 4 deaths in 1964 were infants. A 7-month-old baby with a birth weight of 4 lb. 6 oz. had failed to thrive and died suddenly at home. A 5-month-old child died of gastroenteritis and dehydration. An 8-month-old infant died of what was believed to be pneumonia. A 24-year-old Eskimo woman died "in an epileptic seizure." The last two deaths were at a time when the community was without a nurse.

Baker Lake. By far the largest settlement visited, there were 509 Eskimos in Baker Lake. Of these, an estimated 150 were still living "on the land," coming into the settlement for supplies and visits. The white population numbered 82. At least 80% of the Eskimos were on welfare.

The nursing station was similar to the Coral Harbour station and was capably staffed by 2



Fig. 2. An Eskimo carver and his family in their one-room dwelling in Chesterfield Inlet



Fig. 3. Baker Lake Eskimo children and their response to the pediatrician

sisters (siblings). Housing, vitamin supplementation, and immunization also were more or less as described at Coral Harbour. The diet of the Eskimos, in the main, was bannock and canned meat bought at the Hudson Bay store, supplemented by caribou and some fish.

Of 51 infants and preschool children examined in one day (Fig. 3), 13 were found to have purulent ears and 1 had pneumonia. Another day, 74 children were seen; 10 had purulent ears, 1 had a congenital heart lesion, and 1, a suspected congenital heart lesion.

There were no severe epidemics in 1964, although a wave of respiratory infections struck the community in the spring and again in November and December. There were 30 births in the year, with no stillbirths or neonatal deaths. The 2 deaths were a 31-year-old suicide and a 2-year-old child who suddenly developed a "bloated abdomen" and died "on the land," far from any possible medical aid.

Baker Lake was the one community where I was able to see and visit igloos, the fast-disappearing traditional winter snow houses of the Eskimo. One visit is enough to explain why the Eskimos prefer even the one-room prefabricated oil-heated homes.

(Continued on page 67)



Fig. 4. The one-room prefabricated home found in Whale Cove but typical of housing in this area

An Integrated Approach to Cleft Lip and Palate

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THERE CAN BE few more disturbing experiences to a young mother than bearing a child with the deformity of cleft lip and palate. It is essential that skilled and accurate counseling be available to her shortly after the birth of such a child in order that she may adjust herself to the problem and be in a position to face her friends and relatives in as well informed and adjusted a manner as possible. She will need to appreciate both the immediate problems involved in feeding such a child, the problems involved in the surgical repair of the condition, and the implications of the underlying deformity of the jaws in the subsequent development of teeth and speech. It is for these reasons that The Winnipeg Children's Hospital decided on the formation of a cleft palate team so that parents of these children had one group with whom they could deal. The aim was to help free them from contradictory opinions, and from the worry and distress caused by ignorance and confusion. It would provide the assured knowledge that such methods and resources as do exist for the correction of the deformity were readily available to them as often as they were required.

The reported incidence of cleft lip and palate varies according to the way in which the statistics have been compiled but it would appear likely that in most countries of the Western World about 1 child in 700 has cleft lip, cleft palate, or both. In the Province of Manitoba, therefore, with approximately 20,000 live births a year, we expect to see 30 new cases each year.

CLASSIFICATION

The cleft lip and palate deformity occurs early in intrauterine life and is established by the end of the first trimester of pregnancy. The lip and the anterior portion of the hard palate which extends back as far as the definite incisive foramen in the adult is termed the primary palate and is formed between the fifth and seventh week of intrauterine life. Tondury, Veau, and Stark[1–3] have pointed out that this formation does not, as was previously described, occur by the growing together and fusion of the various processes around the primitive stomatodaeum but rather by the penetration of mesoderm between ectodermal and entodermal layers. A cleft of the primary palate represents not a failure of fusion but a failure of mesodermal penetration, with subsequent epithelial breakdown in the line of the cleft. The portion of the hard and soft palate lying posterior to the incisive foramen is formed in a completely different manner. At the seventh or eighth week of intrauterine life 2 horizontal shelves appear growing out from the maxillary process and hanging down alongside the tongue. As further growth and development occur these palatal shelves come to lie above the tongue and grow toward the midline. Fusion occurs progressively from before backward between the eighth and twelfth week of intrauterine life. That portion of the hard and soft palate formed from these shelves is termed the secondary palate. This recognition of the formation of the primary and secondary palates as embryological processes distinct both in time and nature affords our basis for classification of the cleft lip and palate deformities.[4] There may be either a cleft of part or all of the primary palate alone, part or whole of the secondary palate alone, or clefts of the primary and secondary palate together. This embryological classification is shown in greater detail in Figure 1.

AIMS OF TREATMENT

The aims of treatment in cleft lip and palate are to make the child look, speak, and eat well, and, if one had to choose one of these over the others, it would be to make the child speak well. It is possible for a person to be quite markedly disfigured and still lead a relatively satisfactory life,

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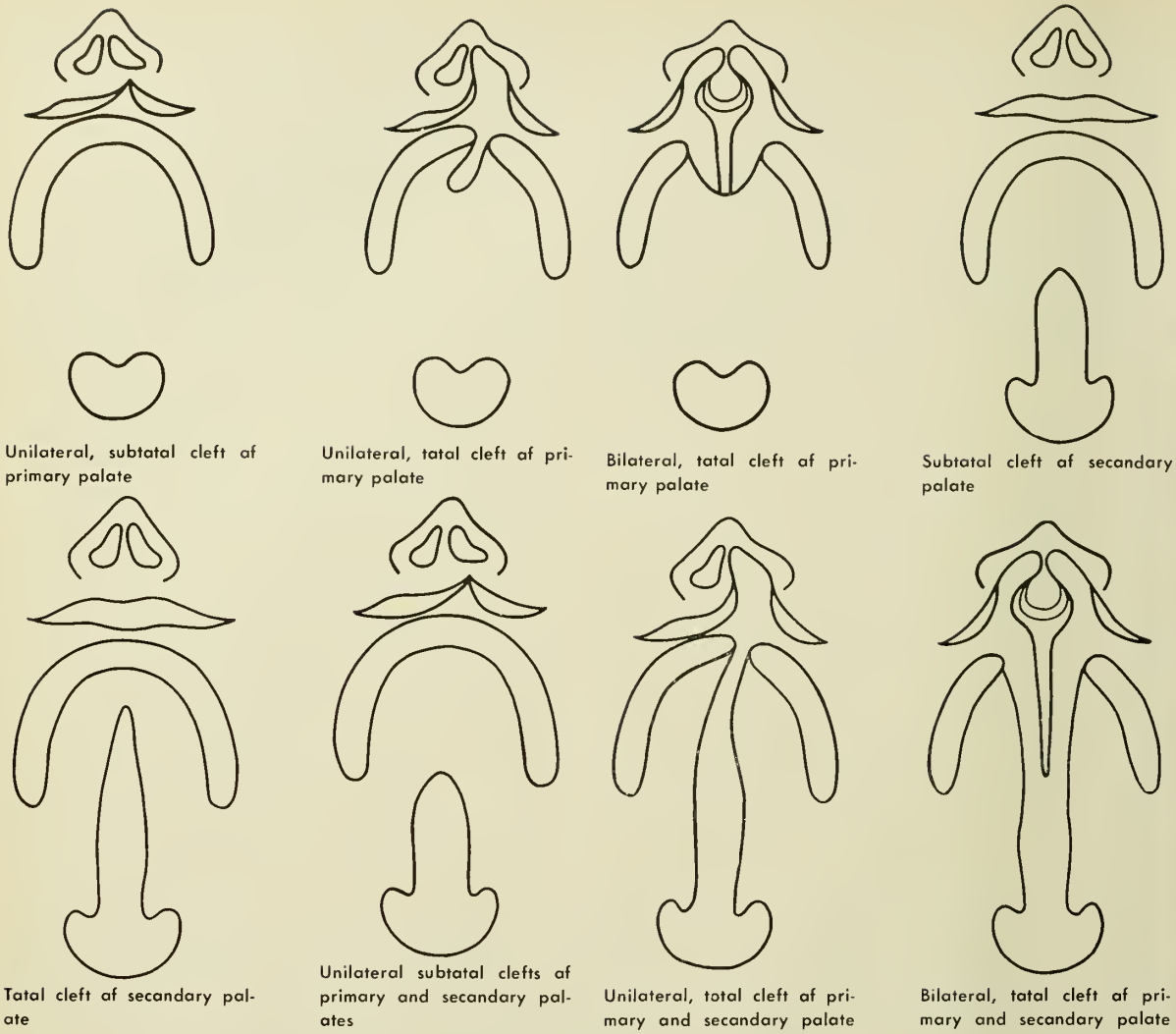


Fig. 1. Classification of cleft lip and cleft palate

but this is not so if speech is so unformed that he cannot communicate with his neighbors. He has then virtually no chance of leading a satisfactory existence and it is for this reason that 2 of the most important members of the cleft palate team are the speech therapist and the otolaryngologist who assesses and deals with any deafness which may interfere with the learning of speech patterns. Making the child look well involves, first of all, a primary closure of the lip defect. Later there may be various plastic surgical procedures to correct the associated deformity of the nostril or of the nose and lip itself. Making the child eat well depends upon the dental surgeon and orthodontist, also essential members of the team. The pedodontist must correct the child's dental caries. The orthodontist must, when the permanent teeth have erupted, institute a program of orthodontia to correct the alignment of the teeth in the malformed maxilla. The prosthodontist may well have to cooperate to replace missing or supernumerary teeth. In addition, the formation of an active team naturally leads to the accumulation of better patient records and the institution of research projects into speech pathology, bone growth, swallowing patterns, and the assessment of surgical procedures so that treatment can constantly be reassessed and improved. In the following short papers, my colleagues have attempted to present some of the procedures necessary for accomplishing these aims.

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Cleft Lip

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Unilateral defects of the cleft lip range from a simple notching of the vermilion to a complete wide defect involving the whole of the lip and alveolus. There is a basic soft-tissue shortage of the lip and asymmetry of the nasal tip and ala. The affected ala is splayed and flattened and the columella is pulled to the normal side. The alveolar defect may consist of a minor notch only, but usually there is wide separation, with a degree of rotation of the medial and lateral segments.

Bilateral defects may also be incomplete, but more generally are complete. The central maxillary segment, consisting of premaxilla and its prolabial covering of skin and vermilion, hinges anteriorly on its vomerine attachment, as it is unrestrained by the lateral maxillary segments. The nasal columella is invariably short and, in extreme cases, the whole central complex may protrude abruptly from the tip of the nose. Rarely a central cleft is encountered.

CORRECTION OF THE DEFECT

Surgical correction in most centers is deferred until the baby is between 6 and 12 weeks of age. Full pediatric assessment is necessary to exclude concomitant congenital defects. Preoperative requirements are a thriving child with a hemaglobin of at least 10 gm. percent and no other ailment, particularly upper respiratory infection. Transfusion at surgery is unnecessary if preoperative anemia has been corrected. Bleeding and clotting time is checked.

Surgery at two days, or shortly after, is advocated by some surgeons, notably Stark,[1] using local anesthetic and sedation. It is generally felt, however, that the older baby is better able to withstand even the smallest blood loss. Surgery is easier on the larger lip, and breast feeding is not interrupted.

In spite of impaired sucking ability, feeding before repair is seldom a problem. It is facilitated by using a soft nipple with an enlarged hole. An aseptic syringe with attached soft rubber catheter may be used, with care, to prevent aspiration.

REPAIR OF THE UNILATERAL CLEFT

In this center, the triangular flap method, popularized by Tennison,[2] and modified by Hagerty[3] and Randall,[4] is most commonly used. It appears to give the most consistently good results, especially in the hands of trainee residents. The methods of LeMesurier[5,6] and Millard[7] are also used.

In the triangular flap method, a triangle of tissue from the cleft side is introduced into a recess which is cut into the uncleft side. The Millard method is essentially a Z-plasty. The central limb is the cleft, the superior limb a cut which runs below the cleft ala, and the inferior limb a cut which extends across the philtrum on the normal side.

The objective of the repair is reconstitution of the orbicularis oris which acts as an orthodontic band and tends to align the maxillary segments. Lip length should be equal on both sides, and the scar should be broken up into various limbs to prevent the contracture of a straight-line scar. A degree of outthrust is imparted to the lip by the introduction of tissue from the cleft side. Accurate vermilion approximation is necessary to prevent a whistle deformity, and a symmetrical cupid's bow is formed.

The nasal floor and the alveolar defect should be repaired, the columella straightened, and the alars matched without buckling.

Preoperative maxillary orthopedics using orthodontic appliances has been suggested by McNeil[8] and Burston[9] in order to bring the alveolar segments into better alignment. These methods are promising.

REPAIR OF THE BILATERAL CLEFT

The bilateral cleft is repaired in one operation by straight vertical cuts on either side of the prolabial tissue on the lateral sides of the clefts.

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Fig. 1. [*Top*] Anteroposterior unilateral view of complete cleft of lip. [*Bottom*] Anteroposterior bilateral view of complete cleft of lip

Undermining of the skin and the mucosa is carried out and the various layers are carefully sutured. The vermilion on either side is joined medially on the inferior aspect of the prolabium, and the prolabial vermilion is reflected superiorly to deepen the alveolabial sulcus. Lateral skin flaps are not inserted on the inferior aspect of the prolabium, however short this may be, as there is accelerated prolabial growth following repair, and the additional length contributed by these flaps is not necessary. Set back of the premaxilla is infrequently required as the tension exerted upon it by the reconstituted oral sphincter mechanism is sufficient to retrodisplace the premaxilla. Only in the worst deformity, where tension on the repaired lip, stretched over a very protuberant premaxilla, would be too great, is the surgical displacement of the premaxilla considered. In these cases a prevomerine resection is done, and the premaxilla is set back and pinned to the vomerine bone.[10] Formal repair of the cleft lip is deferred for six weeks.

POSTOPERATIVE CARE

The lip is kept free of crusting by application of an antibiotic cream to the suture line. A Logan's bow is applied to minimize tension. Cardboard cylinder restraints are bandaged to the arms. Careful suctioning of the nose and pharynx is carried out to prevent airway obstruction. Formula is commenced as soon as possible, and for a few days it is given by an aseptic syringe followed by sterile water. Sutures are removed early, about the fourth or fifth day, to prevent suture marks and nipple feeding is again resumed. Mild sedation is administered in the early postoperative period. Routine antibiotics are not used.

SECONDARY SURGERY

Secondary surgery on the lip is usually deferred until just before school age. Bad scars may be



Fig. 2. [*Top*] Complete central cleft of lip. [*Bottom*] Central cleft of lip repaired

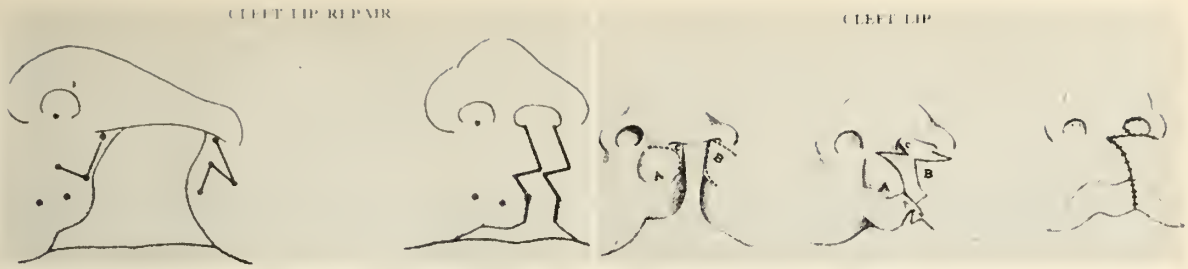


Fig. 3. [*Left*] Triangular flap method. [*Right*] Millard method

revised and a small Z-plasty is usually incorporated to prevent subsequent scar contracture. Step deformities of the mucocutaneous junction are corrected by Z-plasty. Asymmetry of the cupid's bow is corrected by bringing the lower peak up to the level of the higher peak by simple excision of skin at this mucocutaneous junction.

A tight columella causing a pull on the tip of the nose is a common defect following bilateral lip repairs and this is corrected most frequently by a V-Y advancement from the lip.

A tight upper lip which is frequently accompanied by a relative excess of lower lip tissue is corrected by a lip switch from the lower lip.

Alveolar fistula is repaired by overlapping bilateral rotation mucoperiosteal flaps from the alveolar labial sulcus on either side of the fistula.

Surgery on the nose which is frequently deformed is deferred until age 16, when nasal growth has ceased, and usually takes the form of a rhinoplasty. Minor alar adjustment may be done before this time.

Fig. 4. [*Left*] Preoperative view of bilateral cleft lip. [*Right*] Immediate postoperative view of bilateral cleft lip



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The Treatment of Cleft Palate

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THE FIRST SUCCESSFUL repair of a cleft of the soft palate is credited to Le Monnier, a French dentist, in 1764. It was not until seventy years later, in 1834, that the hard and soft palate were closed by Dieffenbach. Since then great progress has been made in perfecting the repair.

The objectives of surgery are to close the palatal defect to produce normal speech, to prevent the soiling of respiratory mucosa and eustachian orifices, and to allow for normal maxillary and dental development. Most surgeons now repair cleft palate when the child is between the ages of 12 and 18 months. It has been shown statistically that better speech is obtained if the cleft palate is closed before the child learns to talk. If the surgery is performed with careful atraumatic technique, no significant growth disturbances are observed.

There are several well-recognized methods of repairing cleft palates. Langerback advocated pairing of the margins of the defect with lateral relaxing incisions along the alveolus undermining the mucoperiosteal flaps and closure of the nasal and oral mucosa into layers.

Wardill described V-Y closure, where mucoperiosteal flaps are raised on either side of the cleft. The nasal mucosa and the aponeurosis of the palate are transected transversely at the posterior border of the hard palate. The greater palatine vessels are freed from their foramen and the palate is "pushed back" with the cleft closed in layers. Approximately 1 cm. of palatal length can be gained by this procedure.

Seventy-five percent of all children with repaired cleft palates achieve good speech. Causes of poor speech in the remaining 25% include: mental deficiency, hearing loss, late repair, postoperative infection, and failure to obtain a soft palate that will affect velopharyngeal closure.

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SECONDARY OPERATIONS

Children with incompetent velopharyngeal closure usually fall into one of two categories: [1] a relatively immobile, short scarred soft palate with a deep nasopharynx and [2] a satisfactory mobile soft palate which is unable to close off a deep nasal pharynx.

In group 1, where the soft palate is relatively immobile, a pharyngoplasty of the Rosenthal type is frequently beneficial. This consists of a superior- or inferior-based flap of the posterior pharyngeal wall being attached to the soft palate and thereby partially closing the nasopharynx.



Fig. 1. Cleft palate before operation



Fig. 2. Cleft palate after operation

The site from which the pharyngeal flap is taken is closed, further reducing the nasopharynx by narrowing the area.

Cases of poor speech which fall into group 2, that is, a mobile soft palate but a very deep nasopharynx, frequently benefit from operations which build up prominences of the posterior pharyngeal wall with which the soft palate can articulate. The insertion of bone cartilage or foreign material into the retropharyngeal space at an appropriate level often accomplishes this. Hynes describes the use of local pharyngeal mucomuscular flaps to build a ridge of tissue on the posterior pharyngeal wall and this procedure has met with good success in properly selected cases.

SUMMARY

Children with cleft palates should have them repaired before they begin to speak, somewhere near the age of 12 months. Seventy-five percent of children with repaired cleft palates obtain good speech. The majority of the remaining 25% can usually be improved by pharyngoplasties. Primary and secondary operations on adults with cleft palates frequently produce a significant improvement in their speech; hence, they should not be considered too old for surgery.

Continued from page 45

NEONATAL HYPOGLYCEMIA: A SIX-YEAR EXPERIENCE

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The Role of the Speech Therapist on the Cleft Palate Team

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One very important reason for the surgical repair or obturation of the palatal cleft is to provide adequate structures for speech. The duties of the speech therapist on the team are: (1) to assess the adequacy of both the speech and hearing of each individual presented to the team, and following sueh assessment, (2) to preseribe an appropriate program of treatment, if warranted.

The clinical assessment of speech made by the speech therapist begins with a systematic study of the patient's speeeh organs at rest and in function. Additional observation is concerned with the degree of velopharyngeal ocelusion. Particular note is made of any constriction of the nares, a positive indicator of either some degree of velopharyngeal incompetence, or the retention of a habit developed at an earlier time when ineompetence did exist.

The speech therapist wishes to obtain a detailed deseription and evaluation of each individual's articulation. An articulation test is commonly used to evaluate one's ability to produce a wide rangc of speech sounds in the initial, final, and medial positions of isolated words and in conversational speech.

Thorough investigation of the hearing status has as its purpose identifying any hearing loss which would interfere with acquisition of speech, vocabulary, language, or educational achievement. The middle ear infection so common in cleft palate children begins early and, even with the best otological care, frequently reduces auditory acuity sufficiently to interfere with speech and language learning.

If surgery has been successful, the decision to prescribe speech therapy may be postponed until the child is 3½ or 4 years of age. Such a deeision is made only if adequate progress is not occurring spontaneously.

The goals of speeeh therapy for each person with a cleft palate are: (1) to reduee the amount of nasal emission, (2) to reduee the amount of hypernasality in the voice, and (3) to improve the aceuracy of speech articulation.

The patient must learn compensatory tongue, lip, and palatal movements which will eventually serve to eliminate the hypernasality and to correct the many sound distortions, thus improving intelligibility of speeeh.

Velopharyngeal incompetenee is undoubtedly the principal faetor in accounting for articulation errors and hypernasality. But not infrequently the presence of hypernasality in the eleft palate person is a product of the total manner of speeeh production. When initiating a program of speech therapy, it is important to change any poor habits whieh will result in the greatest and quickest ehange in the total speech act. Therefore, the initial attack should be on the defects of articulation. Improvement in artieulation will at the same time bring about improvement in resonation, thus resulting in greater intelligibility of speech. As the oral movements and postures of syllable production are normalized, much of the hypernasality disappears. An immediate and favorable change in speech production is encouraging to both patient and therapist.

However, the speech therapist needs to be concerned with more than articulation and voiee disorders. If the rehabilitation program for children with congenital elefts is to be adequate, the goals for therapy must include aural rehabilitation. Auditory training must be part of the therapeutic program, as the reduction of hypernasality and the correction of misarticulated speech sounds is dependent upon sensitive auditory self-monitoring of speech.

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Within certain limits, the speech therapist does her utmost to help the individual with a cleft palate to speak as normally as possible. The therapist thinks of the goals of therapy in terms of improvement. She must be aware that she cannot make speech perfect but can attempt to make speech better and, in turn, make the individual happier.

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Continued from page 50

STRABISMUS: A REVIEW OF 100 SURGICAL CASES

Howard Reed, M.B., and Lillian Bigourdan, A.A.C.O.

cosmetically straight. The services of orthoptists are used more and more for the following: [1] estimation of vision, [2] measurement of the angle of deviation, [3] supervision of occlusion until the vision of both eyes is equal, [4] assessment of the degree of binocular vision during treatment, and [5] follow-up.

Only 16 children were considered to be completely cured, that is, the eyes were straight and the child had normal stereoscopic vision. The eyes of 74 children were cosmetically straight, and many of these had some degree of fusion but no stereoscopic vision. In 10 children, the results were cosmetically poor.

VISUAL RESULTS

Of the 64 children with previously convergent strabismus, 43 had 20/30 or better vision in both eyes (Table 5). In 19, the vision of one eye was less than 20/30, and in 2, the vision could not be determined because of lack of cooperation. The 10 children who had previously divergent strabismus all had 20/30 or better vision in each eye, with fusion but no stereopsis.

DISCUSSION

A cure rate of 16% is low, but these children were a selected group. All were staff children and, as can be seen in Table 2, one-third of them first attended the clinic long after onset of the squint —past the age at which a good result can be obtained. Moreover, due to socioeconomic conditions, cooperation was poor and parents often failed to insist on the constant wearing of glasses. Occlusion was not carried out adequately, and return appointments were not kept. This occurs less in private practice when most children are seen during the second and third years of life. In a previous study of private patients,[1] about one-third obtained a complete cure. Thus, it is probable that, as time goes by, more children will be seen as soon as the squint appears and the incidence of complete cure will become still higher. In almost all cases in which a complete cure cannot be obtained, it is possible to make the eyes cosmetically straight with equal vision in each eye.

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Dental Aspects of the Cleft Lip and Palate

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The postnatal growth and development of the orofacial and pharyngeal regions is of great importance to diagnosis and treatment planning for individuals with cleft lip and cleft palate, or both. Growth and development must be considered in terms of both morphologic changes in the skeletal and soft tissue structures involved and also in terms of the development of the functional aspects of the stomatognathic system.

The inherited postnatal growth pattern of the face in a cleft palate patient does not seem to differ from that of a normal face, insofar that growth proceeds regularly and in harmony with the basic deviations brought about by the cleft. The suggestion that patients with clefts develop a characteristic type of skeletal and dental deformity would seem to be unjustified, in that any genetically determined skeletal deviation or dental malocclusion can develop in cleft patients just as in normal subjects. The cleft deformities are merely superimposed upon the individual's normal inherited pattern of growth. In short, it is found that the orofacial skeletal and dental deformities associated with clefts are of infinite variety and complexity and therefore pose many problems to the diagnostician.

The ability to diagnose, follow, and predict growth changes from birth in a cleft subject would appear to be of major importance in the overall rehabilitation of such patients. Without such knowledge, treatment must be empirical and often questionable in terms of prognosis. With the team approach to cleft palate rehabilitation, the possibility of integrating the various surgical, dental, orthodontic, and speech treatment procedures in terms of growth and development becomes possible. If craniofacial skeletal development, dental development, and velopharyngeal development and function can be assessed accurately, then treatment procedures can be timed to suit the individual patient.

The growth of the craniofacial skeletal structures can be assessed and followed using cephalometric radiography. This technique consists of taking lateral and frontal head radiographs using a head orienting device. The radiographs are taken at intervals from birth. Measurements can be made on tracings of these radiographs and can be used to determine objectively the growth progress of the skeletal structures before and after operative procedures. Cephalometric radiography assumes an important role in assessing the results of orthodontic therapy which will often be required later in the rehabilitative program.

A diagnostic aid which is becoming increasingly useful for both research and diagnosis is cinefluorography. Standardized cinefluorographs of the orophyarngeal region can be used to assess the postnatal functional aspects of the velopharyngeal mechanism, the lips, tongue, and mandible, during such activities as speech, deglutition, and mastication. If the cinefluorographic recordings are made on film, it is possible to analyze the films in a frame by frame manner and objectively assess the movement patterns. When synchronized with speech recordings and pressure measuring, or air flow devices, a comprehensive, physiologic, diagnostic work-up can be made of the functional impairment associated with many cleft cases. A combination of the above mentioned data and the information available from cephalometric radiography and serial plaster models of the dental occlusion will probably prove to be of increasing importance in terms of cleft palate diagnosis and treatment planning.

Research has demonstrated the fact that in most instances surgical procedures alone are inadequate for complete cleft rehabilitation. More

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than surgical repair is needed if correct oral function is to be achieved. There appears to be a correlation between the timing and type of surgery and the oral deformity which is noted subsequently. Surgery which is carried out early and is traumatic seems to give the greatest degree of deformity. However, with the advent of better surgical procedures, this problem appears to be somewhat overcome. An example of the dental deformity associated with repaired cleft lip and palate is in the unilateral cleft of the lip and hard palate in which the teeth on the side of the cleft are usually in a lingual cross-bite relationship with the opposing lower teeth (Fig. 1). In bilateral clefts, the premaxilla is sometimes displaced forward or, as the result of a "tight" lip repair, is forced lingually (Fig. 2). The maxillary incisor teeth in these cases are usually badly malposed and tipped. Teeth in the region of a cleft may be abnormal in number or shape. Congenital absence of teeth is common and supernumerary teeth also occur. Teeth may be abnormal in shape or, on occasion, they may be "twinned." In many of these instances it will not be possible to obtain a perfect occlusion of the teeth by orthodontic means. Teeth may require to be replaced prosthetically and permanent fixation of bony segments may be required using either tooth-borne fixed bridgework or bone grafting.

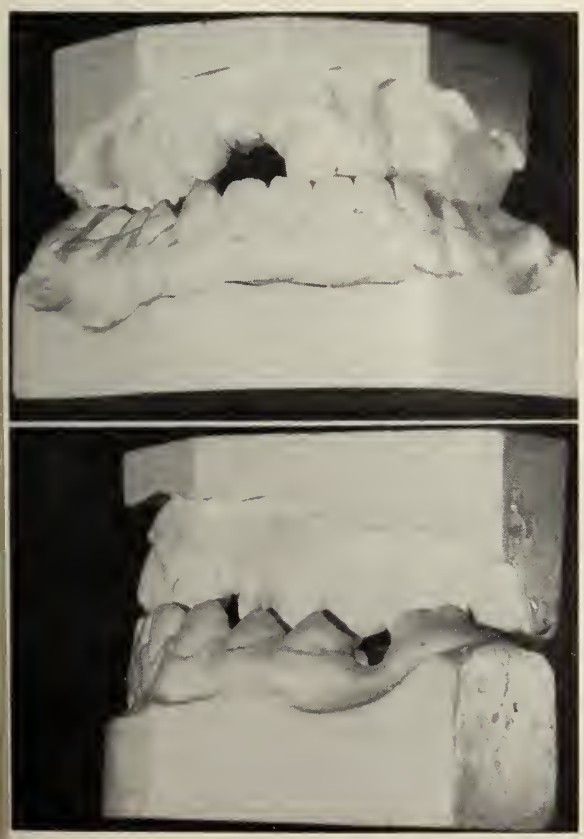


Fig. 1. Showing dental deformity associated with unilateral cleft of lip and palate

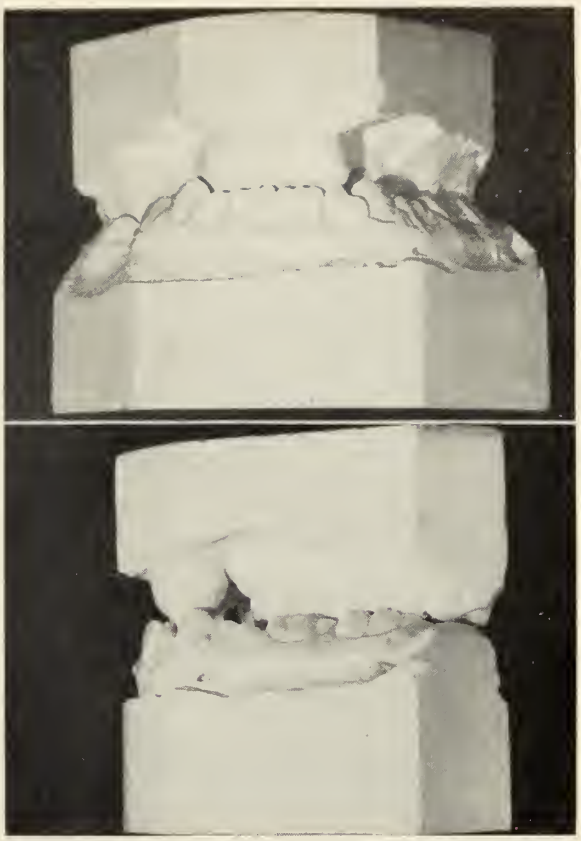


Fig. 2. Showing dental malocclusion associated with bilateral cleft of lip and palate. Note collapsed buccal segments and incisor cross-bite relationship.

A consideration of paramount importance to the orthodontist, surgeon, speech therapist, and prosthodontist alike is the maintenance by the general dental practitioner of the teeth themselves. Dental conservative work must be routine along with oral hygiene instruction and the institution of a program of caries prevention. If not, teeth will be lost due to the ravages of dental caries, orthodontic treatment will be impossible, and the later placement of prosthetic speech bulbs and such will often be impractical.

Every case must be assessed in relationship to the nature of the original deformity, the growth potential and progress, and the functional performance; a brief general outline of ideal orthodontic therapy timing may not be out of place. Within the first few weeks of life, a brief period of orthopedic bony segment molding using small plastic orthodontic soft tissue-borne appliances may be indicated. This may assist the surgeon to orient the structures better at the time of the

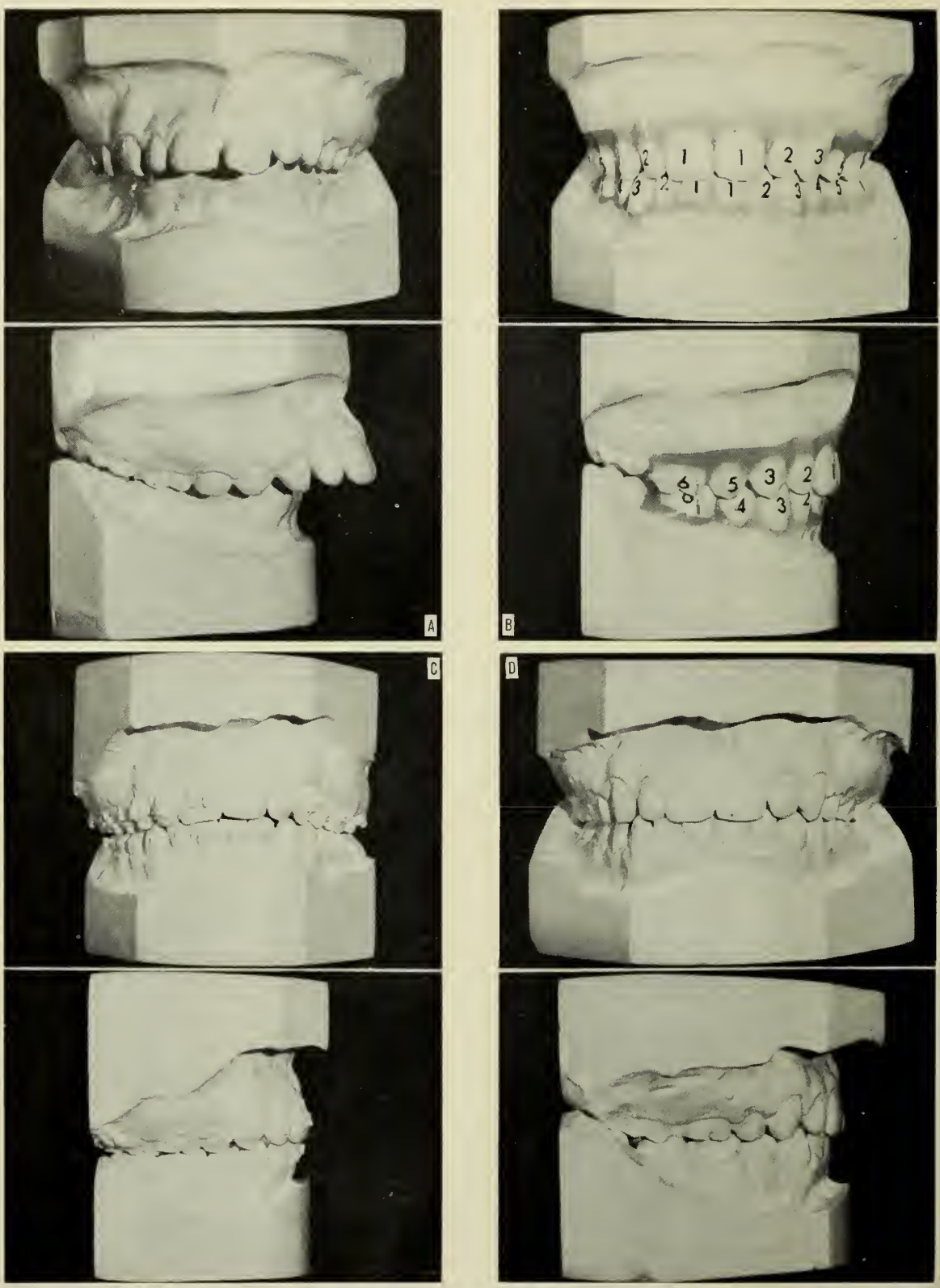


Fig. 3. Routine orthodontic treatment following repair of unilateral cleft lip. Note that despite cleft lip, a severe Class II malocclusion has developed, demonstrating that cleft deformity is merely superimposed upon individual's genetically determined pattern of facial growth (A) before treatment, (B) diagnostic wax set-up, (C) during treatment (note orthodontic bands on teeth), and (D) two years after completion of treatment.

initial lip repair. Later, during the subsequent year or two, a similar period of treatment may be advantageous before the palate is closed.

The timing of successive and more active orthodontic treatment will depend upon the cooperation of the patient, the stage of dental development, and the timing of any remaining surgical revisions. Usually treatment to expand the collapsed lateral segments or to correct the incisor teeth will be started between 7 and 9 years of age. Once the preliminary work is completed, it will often be necessary to place a small removable retaining appliance in the mouth until such time as most of the remaining permanent teeth (excluding 3rd molars) have erupted at 11 to 12 years of age. At this time, final orthodontic correction of the permanent teeth will be carried out and may be followed by final surgical revisions or permanent splinting procedures (Fig. 3). The prolonged nature of orthodontic treatment may interfere with such rehabilitative procedures as speech therapy, and often a compromise must be reached to assure the child the best overall rehabilitative care.

Continued from page 54

SOME OBSERVATIONS ON ESKIMO HEALTH

Fishel J. Coodin, M.D., C.M.

Whale Cove. This small community located 70 miles south of Rankin Inlet has 181 Eskimos and 19 whites. Hunting and fishing are poor in the area, although a struggling cooperative store and a fish cannery provide employment to a few Eskimos. Approximately 80 percent of the Eskimos were receiving welfare. Housing was in the prefabricated homes (Fig. 4), and health matters were handled by a lay dispenser. Food was purchased largely from the cooperative store. There were no deaths in this community during 1964.

Eskimo Point. In this community of 407 Eskimos and about 28 whites, 80% of the Eskimos were receiving welfare. The nursing station was as described at Coral Harbour, and it was staffed by a single, highly competent nurse. Vitamin supplementation was as described at Coral Harbour. Food was chiefly purchased from the Hudson Bay store.

Of 48 children examined in one day, 9 had infected ears. The water supply was atrocious; it was considered necessary to immunize against typhoid. Dysentery was rampant throughout the summer and ended only in October. It was only a couple of years since this community had been hit by an acute epidemic of tuberculosis, with about 80 evacuees and 6 deaths. Two adults and 2 children died of dysentery, 3 adults died in a boating accident, and a 6-month-old infant died of a respiratory infection in 1964.

Churchill. Located in northern Manitoba, Churchill is an indescribable seaport town of 7,000 inhabitants On the departure of the military, a military hospital recently was converted into a community hospital. The staffing problems were acute. All the Eskimos from the areas described were funneled through this unit and then transferred south to Winnipeg when necessary. At the time of my visit, the medical staff consisted of 1 physician employed by the Department of National Health and Welfare and 1 physician in private practice.

SUMMARY

Over the last twenty years, the Department of National Health and Welfare has made tremendous progress in providing the Canadian Eskimos with improved medical care. The Eskimo population is increasing at a much faster rate than that of other parts of Canada. These Eskimos are changing from the stone age to the modern world in practically a generation. One cannot help but recognize that methods are desperately needed to help them out of their educational, cultural, and economic plight. This done, their health problems would automatically be greatly reduced. However, diet and other preventive measures, together with emergency medical services, will always be necessary, although difficult to manage, as long as they continue to live in such remote areas and in such small groups.

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The Child Development Clinic of The Children's Hospital and Its Method of Collaborative Treatment

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THIS CLINIC, which has been functioning for just over two years, was set up according to a plan prepared at the request of the Psychiatric Services Committee of the Welfare Council of greater Winnipeg in February of 1958.[1] It is an insured service under the Manitoba Hospital Services plan. This means that any Manitoba preschool child not yet 7 years old, who is referred by the family physician or a physician in a public clinic, will be seen by appropriate members of the staff on an out-patient basis without charge to the parents and with full reports to the referring physician and appropriate health or welfare agency. The professional staff includes a social worker, a psychologist, a nursery teacher and her assistant, and 3 speech therapists. The 2 medical members are pediatricians with special training in child development, the director also being a psychiatrist.

The clinic is prepared to attempt to deal with any problem of delay or deviation in development. This may be in the motor sphere, in the sphere of communication, a behavior abnormality, an intellectual deficit, or an abnormality existing only in the mind of the parent. To date, there has been little intensive psychotherapy, but wherever possible, continuing interest is maintained by regular contacts with the social worker or, on occasion, by attendance of the child for a varying period in the development clinic nursery school.

The nursery school accommodates no more than 10 children at a session, 3-year-olds in the morning and 4-year-olds in the afternoon. Each of these groups has a nucleus of about five more or less normal children, who are usually the children of medical residents in specialty training in the area or of staff members of the hospital. Other children are brought in for diagnostic purposes (which invariably involves some therapy) or frankly with a therapeutic aim. The experience with one of these children will form the second part of this article.

TEACHING

The clinic members are all engaged in teaching. This may entail demonstrations to undergraduate medical students, learning by association of senior residents in psychiatry and pediatrics, lectures to nurses or social workers, or periods of observation and discussion of children in the nursery school.

During the first year of operation 607 different patients were seen by the 2 members of the medical staff. The largest single group (180) was referred to Dr. McRae by children's aid societies and other welfare agencies, many of them for preadoptive examinations. Service here involves helping adopting or foster parents with the children they choose to take. We give them as complete information as possible without necessarily advising strongly that a child may or may not be "adoptable" (which we consider a non-medical adjective).[2]

ANNE BELL *is clinic psychologist;* SANDRA LOWE *and* DOROTHY KEITH *are former staff members in social work and nursery education, respectively;* K. N. MCRAE *is assistant director and assistant professor of pediatrics; and* W. WALLACE GRANT *is director and associate professor of pediatrics, Children's Hospital Child Development Clinic and University of Manitoba, Winnipeg.*

MENTAL RETARDATION

We have specifically avoided having the clinic labeled as a mental retardation facility, since we are interested in children with a great variety of problems who are not necessarily mentally retarded. Furthermore, many parents will bring their children for early help, with the difficulties they present, if a clinic has no such emotionally charged label. However, when we do see children with mental retardation we make an effort to determine etiology, to assist with current problems (in child behavior and parental attitude), and to offer continuing assistance until the child is ready for school. Service is then carried on by child guidance clinics or other facilities set up for school-age children.

Specific forms of mental retardation seen in the first year included phenylketonuria in 5 children. Among these was the first newborn infant diagnosed in this area by the Guthrie technique. The local Manitoba experience with 26 phenylketonurics has been described in another paper.[3] Phenylketonurics are not regularly followed in the clinic. The attending pediatrician requests our help when it seems needed. The Provincial Health Department requires clinic corroboration of diagnosis before providing diet material without charge to the parents. A Manitoba registry of known phenylketonurics is kept up to date in the clinic.

Nine children with mongolism were seen by one of us (W.G.). In each instance, there were problems other than mental retardation with which we attempted to cope. These included organic hyperactivity in identical twins who had been premature, severe maternal depression, marked paternal rejection, an adjustment reaction involving marked negativism in a 3½-year-old, obesity, and two instances of behavior difficulty which seemed to be related to excessive parental expectation. These children all had chromosome analysis by the cytogenetics laboratory of the hospital (D. I. Vchida, director). Eight were found to have 47 chromosomes (with 21 trisomy), and one had 46 chromosomes and a G/G translocation without similar pattern in the relatives. One of us (K.M.) works as the medical consultant to the department of genetics and has collaborated with them on several papers.[4,5] Dr. McRae's study with Dr. Haworth of neonatal hypoglycemia is reported later in this journal and elsewhere.[6]

We tend to avoid emphasizing I.Q. or D.Q. labels on very young children. Our interest is in etiology and assisting with problems, not in categorizing. The range of intelligence of children seen is indicated by the fact that, of the large group of children referred for particular problems, the estimated I.Q. was 0 to 20 in 4%, 20 to 50 in 12%, 50 to 70 in 18%, 70 to 90 in 23%, 90 to 110 in 36%, and over 110 in 7%. Some of the severest disturbances in behavior with which we have had to contend have been manifested by children with I.Q.s from 120 to 150.

COMMUNICATION DISORDERS

About 5% of the children referred with problems in the first year proved to be deaf. Many of these were part of a larger group referred by the Society for Crippled Children and Adults of Manitoba regarding eligibility for its preschool deaf nursery and treatment program. This group is being analyzed for a more complete presentation. We have been concerned with difficulties in early diagnosis; problems in discrimination between deafness, dysphasia, and psychosis; frequent association with growth failure (30% of our group were 3 percentile or less in height); and very commonly associated difficulties in impulse control and hyperactivity.[7] Many children with language delay are included in the 647 patients examined during the year by the speech department. The clinic staff, and especially Dr. McRae, are cooperating in a pilot preschool program for "aphasic" children under the auspices of the Society for Crippled Children and Adults of Manitoba.

OVERACTIVITY

One-quarter of the "problem patients" appeared to be suffering from some degree of the "hyperkinetic syndrome." One-half of this group had intelligence which was average or better. Commonly (especially if there were evidences of organicity), associated difficulties included speech delay and articulation problems, sleep resistance, clumsiness, and various so-called minimal neurological findings (see case presentation below).

PSYCHOSIS

More than 6% of the children seen by the two medical staff members during the year were frankly psychotic. In almost every instance it was extremely difficult to establish specific etiology. We are impressed with the fact that frequently history and examination suggest an underlying organic abnormality in the child. This difference appears to have led to altered handling by his parents. It often made him the "scapegoat" in the family and may have led him to seek escape in fantasy. Early identification of children with organic behavior disturbances seems especially important if it can lead to modification in handling and perhaps prevent the development of this type of psychosis.

Almost 25% of our patients appear to be suffering from an adjustment reaction of childhood. We often find it difficult to categorize definitely as to whether a problem is specifically a conduct disorder, a habit disorder, or a neurotic trait, since not infrequently a child will appear to have combinations of two or more of these disabilities. We have especial interest in preschool children with sleep resistance[8] and are impressed with the fact that a large number of children we see with stool retention and soiling appear to follow no particular pattern in the development of their disturbance.

CASE REPORT

The complicated nature of the problem, presented by a more or less typical child, is illustrated in the example that follows. This was a recording of a program on Jan. 20, 1966, at regular weekly grand rounds of the hospital medical staff. The way the child was managed emphasizes collaborative effort involving various members of the clinic personnel.

Introduction (Dr. Grant). This child Johnny we call a "kindergarten kickout" because he was, in fact, just that. We received beforehand from his private kindergarten teacher his "expulsion diploma." In it she has listed, after nineteen days with him, 12 reasons why this little boy was inappropriately placed in her tidy setting.

"1] He balked at all routines.

2] He destroyed other children's constructions for no apparent reason, no matter who the individual was or where he was.

3] He had a fleeting attention span and moved from one activity to another without exerting time or thought. He seemed to show improvement with time.

4] He continuously touched, shoved, bit, or poked anyone near him.

5] He continually made grunting and disturbing noises and loved to make loud banging noises.

6] His speech was not clear.

7] He continually moved parts of his body by shaking, jumping, and hopping.

8] When approached by an adult, he responded slightly but didn't show complete comprehension as to why he was approached. He would immediately go on doing what he was approached for.

9] He resisted all commands but seemed to be improving with time.

10] On entering the kindergarten he was unable to manage his clothing—take off or hang up coat or take off or put on shoes. He showed improvement here in time.

11] He seemed happy but elated.

12] He bites his thumb and chews anything he is able to put into his mouth."

With this introduction, I prepared my office as if for battle on the day of his appointment, Oct. 23, 1964, when he was 4 years, 10 months.

He was brought by his maternal grandmother, who will be described by Miss Lowe. This was a large, fat boy who, although not yet 5, had the height of a 7½-year-old and the weight of a 10-year-old. Height was therefore at the 97 percentile, weight was considerably above this. He was left-handed but used the right fairly well. I thought there was questionable up-going toe on plantar stimulation on the right side but no other evidence suggesting neurological abnormality except for the isolated left-handedness. Visual acuity was perhaps down a trifle, and hearing was normal. He was inclined to be distracted by minor stimuli in the environment. When I was examining him he was likely to try to pull open drawers, reach across to spin a chair, and grab other things nearby. This became increasingly a problem as the tasks given to him became more difficult. It was hard for his grandmother to avoid comment or urging, although I asked her many times to take off the pressure. His performance in general was more like that of a child of 4, although there were some successes at the 4½-year level. One got the impression that if he could have kept his attention on the problem at hand and felt less pressed, he would have managed better.

He was the product of the first of about five pregnancies of an unmarried mother, now 24. She reached only grade 7, is presumably rather dull, and has been a source of great concern to her parents since she was small. She has a child born in March 1964 who is with her, another has been placed for adoption, and there were two miscarriages. Mother stayed with her parents until Johnny was about 3, ostensibly caring for him, but, according to the grandmother, giving him very little attention. The maternal grandmother has been providing total care since her daughter left home, and this involved taking the boy with her when she went babysitting during the day. His birth weight was 8 pounds, 12 oz. There was nothing particularly unusual about his early development. In the motor sphere, he pulled himself to stand before ten months. There was some difficulty with toilet training but he now wets the bed only occasionally and has good urine control in the daytime. Feeding presented no problem except for excess intake, and he used a spoon and a fork with reasonable skill. Resistance to going to bed, formerly considerable, was not so now, and he accepted a bedtime of 8 or 9 o'clock, sleeping through the night but getting up at 7 o'clock. Unfortunately, because of space limitations, he slept in the grandparents' room. There was only occasional opportunity for play with other children in the neighborhood.

In the process of the examination he drew a picture of a boy. At 4 years, 10 months you don't expect very much in the way of accuracy and artistic delineation of features. The striking thing was that he drew hair and eyes, a few other miscellaneous features, and long legs, but no boundaries. One wonders if this were the way the child thought of himself and his relationship to his environment, rather than evidence of his limited capacity to reproduce form.

We then suggested to the referring physician that we would be very much interested to see just what change might be produced in this boy in a benign environment, where an effort would be made to encourage him to think better of himself. I noted that these children with overactivity and distractibility are often considered suffering from neurological impairment but that I woudn't be surprised if this became less of a problem as he became more at ease in our nursery setting.

Now I would like Miss Lowe to tell you of her experience with the grandmother.

Miss Lowe (social worker). Johnny lives with his middle-aged grandparents. Thrice married, the grandmother has lived through many trying experiences and

been on the verge of an emotional breakdown several times herself. Her third marriage is a satisfactory but somewhat unusual one. She is a large, rather anxious woman who tends to take on the problems of the universe. She often looks after "stray" teenagers and, while not being too successful with her own, worries about the children of others. The grandfather is a mild little man who lacks both understanding and experience with young children. Johnny's mother, who is now 25, presented severe emotional and behavior problems from early childhood, these becoming more severe in her teens. Ability was limited, and she failed and left school in grade 8. She was irresponsible and eloped from the home several times, necessitating police and juvenile court action. She refused help and training offered and left home finally at 17, thereafter becoming involved with several soldiers whose treatment of her was rather questionable. When pregnant with John, placement was considered but, with her parents' support and their hopes that the baby would help her settle down, she returned home. She neglected John and left home to establish a common-law union which later broke down. The grandmother obtained legal guardianship of the boy when he was 3. The child, however, continued to be exposed periodically to his mother's disturbed and erratic influence.

John at home was initially described by the grandmother as being a very stubborn, headstrong, active little boy. She felt some of his behavior was attributable to his having been scared by his own mother's handling and attitude toward him. In his peer relationships in the block he had learned to fight and seemed to feel the only way one received things was to push people around. His relationships with his two teen-age uncles and 20-year-old aunt in the home were good, although they tended to baby him and foster his dependence on them. He considered the grandfather as his father, but more problems existed here as the grandfather was unable to be consistent in his attitude and frequently lost his patience with the child. While generally the home appeared to be a stable one and the attachment to the child sincere and positive, there was probably a degree of resentment of the child because of the responsibility he imposed.

Subsequent contacts were maintained with the grandmother and problems that came up during the year were discussed with her. The family felt there was a good carry over from his happy nursery experiences and any untoward behavior was disappearing from the home. He took pride in newly acquired skills such as dressing himself. The entire family was cooperating in order that John might attend the nursery school. Working nights, the grandfather brought him ten miles on the bus daily from an eastern suburb and then put in two and one-half hours in the hospital area waiting for him. The teen-age sons began dinner preparations to allow the grandmother to continue working. The influence of his mother's visits was discussed thoroughly with the grandmother, who was able to become more emphatic in discouraging this. Interpretation was offered to help her understand specific behavior John presented. Alternate means of handling were suggested, such as ignoring outbursts rather than becoming upset herself and giving up the rather prevalent pattern of bribing this chubby little boy into doing things by offering a never-ending supply of chocolate bars. I generally feel that the family's expectations of Johnny are realistic and that they are accepting him as he is now.

Miss Keith (nursery teacher). I'll start with a description of Johnny's play behavior, remembering the report that we got from the kindergarten teacher. I think that much of what she said was true. However, our opinions differ in that I would never consider these as reasons for expelling a child from a play situation.

In his play, Johnny used tremendous energy. While he painted, for example, he would slap the brush hard on the paper, rock back and forth, and often move his feet at the same time. He attacked plasticine and clay in much the same fashion, pounding, rolling, and slapping the material on the table. He usually stood to do this so that he would get more force behind the activity. The blocks were initially used simply to stack and pile in large boxes, which were then pushed across the floor. The first afternoon John was in the nursery for regular sessions, he swept every block off the shelf, piled them up, and then began putting them back. To an adult not familiar with the way he operated, it would often appear that his play would explode in meaningless chaos, but we learned that as wild and unorganized as it sometimes appeared, it was constructive and would be finished up and, above all, was completely satisfying to him.

For several weeks John was very much a loner in the nursery group. Gradually he began to play with two of the boys in the normal group. This friendship began to mean so much to him that he became very upset when he couldn't be close to them. For a period, he began to act up. Living up to expectations of behavior was difficult for John in the beginning. His reaction to requests with which he didn't wish to comply was to lie on the floor and pout. This reaction disappeared after the first month but reappeared later when he was having difficulty in his relationship with other children. Ignoring, and sometimes using isolation, proved to be effective in these situations, although his refusal to go home and thus end association with his friends never really was happily solved.

Notable, too, was John's demeanor and appearance when he first joined us. In fact, during our initial contact he visited the nursery while the 4-year-old session was in progress. The children were having juice and cookies at the table and, even with John's strong desire for food, all he could manage to do was to stand inside the door with his head lowered, looking the very picture of a dejected little fellow.

He tended to be without verbal expression when he first came. His means of getting attention was to hit the teacher on the arm and point to the situation or event he wished her to notice. Slowly these disappeared and he began using language, first to communicate wishes and needs, and later he began to play with others. Speech blossomed forth conversationally and as an accompaniment to play.

Likewise, at first he seemed a kind of immobile blob when it came to any kind of self-help. Dressing was indeed a problem because he was so fat his clothes were generally too small for him, and he couldn't realistically handle buttons. He did learn to undress and dress himself, however, and if he couldn't manage he would ask for assistance rather than just waiting for it. It seemed to us that John, despite his size, was a very immature little boy, particularly in social areas.

In the seven months he was with us, I feel he did a lot of growing but needed continued experience of conforming to group rules with realistic guidance. By that I mean avoiding bribery and harsh punishment. He also needed experience in accepting logical consequences of his actions.

Mrs. Bell (psychologist). I saw John in June 1965. This would be at the end of his seven months in the

nursery school. I gave him the Stanford-Binet, Metropolitan Reading Readiness, the Hooper Visual Organization, and the Closure tests. At this time the problem was whether he should stay away from school another year or go on to grade 1. He had a December birthday and where he lives he could enter school in the fall. We were therefore particularly anxious about his readiness for school.

The Stanford-Binet mental age was 5 years, 2 months. He was at this point 5 years, 5 months. The basal age was 4½ years and further credit was scored up to the seventh year. This suggested considerable unevenness in development. The I.Q. was 95. The Metropolitan Reading Readiness score (language usage and so on) was average for his age, so there was some strength in that area. On number readiness he was a very poor risk. He had no concept whatsoever of numbers. Total readiness came to a low-normal score, the same as his Stanford-Binet I.Q. He was at about the nineteenth percentile for children entering grade 1. However, he did show considerable strengths on the perceptual tests. On the Hooper Visual Organization test he correctly managed 20 out of the 30 cards, which was above the average for 6-year-olds. The Closure Test has 10 cards and the average score for 6-year-old school beginners seems to run around 6 or 7 out of the 10. John managed 8 out of the 10, which was exceptionally good for 5 years, 5 months of chronological age.

So he had certain skills in this integrational activity. I suspect that our current study in this field will show this to correlate highly with success in school. I noted, however, his very poor motor skill. No matter how accurately he perceived, he certainly was going to have trouble with the expression when the teacher asked him to hold a pencil or crayon. This I asked him to do and he could scarcely manage to grasp them. Because of his poor motor control, a below average I.Q., and a December birthday, I would normally suggest another year in kindergarten. In view of the strengths he showed, his large size, and the situation in his previous kindergarten, I thought it was likely that he would be better off in grade 1 in his particular school.

Dr. Grant. Mrs. Bell contacted the school by telephone before this child started so that they were aware of our experience with him. As a consequence, I think they managed him somewhat differently than they might otherwise have done.

I saw him on Dec. 17, 1965, and he busied himself in the nursery during my interview with the grandmother. She was mainly pleased about how things were going. In the first month or two of school she'd been disturbed because the boy was very unhappy, was being picked on by the other children, and had continued to gain weight in spite of an appetite-suppressing drug which had been prescribed by the family physician. (Incidentally, he had a chocolate bar in his hand as he was waiting, so we haven't completely convinced her that sweets aren't the best way to demonstrate affection for a child). The teacher modified her methods on the basis of our advice and after discussions with the grandparents. Since then, there's been marked improvement.

He is now in his own bedroom since his aunt has moved out, and this gave him quite a boost. He's getting along much better with his grandfather, and in general seems to have lots of friends. Grandmother commented especially that he even looks forward to school and to such a degree that he wants to rush through his breakfast. This is so even on Saturdays, when she has to tell him that it isn't a school day. Our most recent report from the school is that although the boy tended to be a little unhappy originally and cried if he were pushed to achieve, he certainly had no problem in adjusting to the other children. As has been suggested by the grandmother, he does like school and he gets along well, although it's obvious that he is going to have problems getting into grade 2 next fall.

Summary. What in essence did we do here? What was the problem and how did we approach it? In the first place, this is a child who obviously had a very difficult beginning. I can't imagine a less welcome Christmas present than this boy who was born on December 25 to a mother who was unmarried, who had very little capacity to handle him, and, perhaps unfortunately, refused to have him placed for adoption. The grandmother is an affectionate person, but she has mixed feelings for obvious and understandable reasons. Four other adult relatives, not always in agreement about his handling, made up the household, his vagrant mother upsetting everyone with her periodic arrivals and departures.

The boy himself has certain physical difficulties. He looks like 10 when he's not yet 5, socially he's 3 or less, and intellectually he isn't far off his chronological age. He also has a certain amount of clumsiness which you could say was a neurological problem. On top of all this came the sudden, predictably unsuccessful, placement in an unfamiliar and rigid kindergarten when he was still not yet 5.

We tried to teach him to adapt and adjust in a benign environment where a fair amount of his seeming misbehavior was tolerated. Furthermore, there was advice and support to the grandmother, who we felt was really doing her best. We were most impressed with the degree to which every single member of this family was prepared to put himself out in order to give this opportunity to the boy. At the end of this period, we did have some contact with the school which is continuing. We like to feel that this kindergarten "kickout" may now very well make a good adjustment and not end up as an elementary school "dropout."

If we have helped to make this come about it is through a cooperative effort involving all our staff, the child and his family, and the school to which we graduated him.

CONCLUSION

This has been a partial review of the first year of operation of the Child Development Clinic of The Children's Hospital of Winnipeg. Collaborative effort by several of the staff in relation to one child and the use of the nursery school in his treatment are described.

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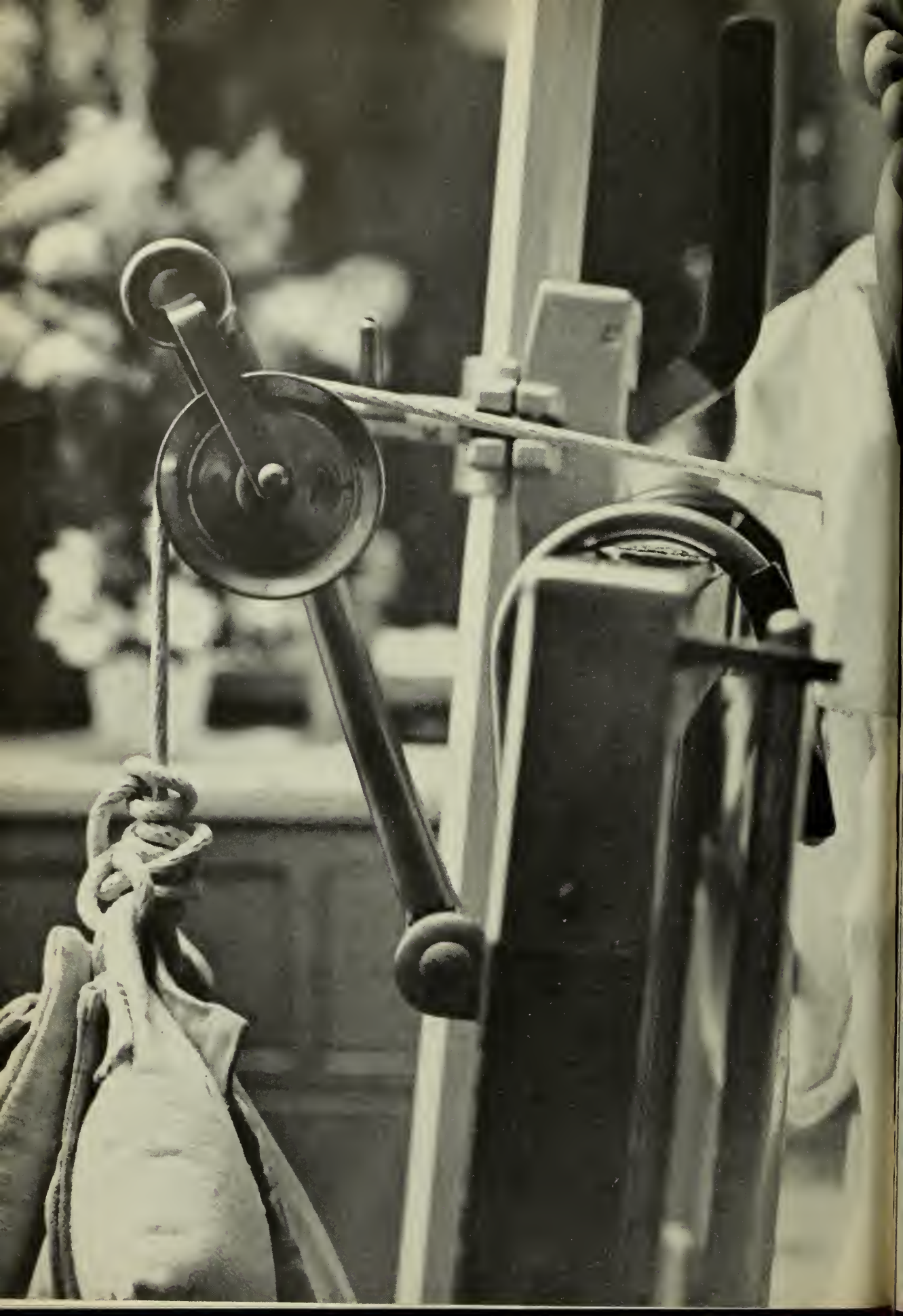
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News Briefs . . .

North Dakota

DR. WILLIAM T. POWERS has succeeded the late Dr. Charles Graham as Grand Forks city health officer.

* * * *

DR. C. J. KLEIN, Valley City, has been installed as president of the American Academy of General Practice, North Dakota chapter.

* * * *

DR. NEVILLE JONES, formerly of London, England, has opened a practice at the Towner Medical Clinic in Towner.

* * * *

DR. E. A. HAUNZ of Grand Forks was honored at a meeting of the North Dakota Institute on Rehabilitation, Labor, and Workmen's Compensation for his assistance to handicapped persons in the state.

* * * *

DR. SAID GHATAN, of Michigan, N.D., a native of Teheran, Iran, has joined practice with Drs. Gordon and Jane Magill in Fargo.

* * * *

DR. MILTON O. BERG has been appointed city health officer for Valley City. He succeeds the late Dr. A. C. Macdonald.

Minnesota

THE MINNEAPOLIS CLINIC OF PSYCHIATRY AND NEUROLOGY has announced a number of new staff associations. They include: Dr. Paul W. Bransford, adolescent and child psychiatry; Drs. Thomas H. Davis and John M. McKelvey, neurology; Drs. Thomas P. Burton, Bruce H. Hiller, and Glenn M. Lewis, Jr., psychiatry; Nicholas Borrelli, Ph.D., psychology; and Norman J. Segal M.S.W., social work.

* * * *

UNITED HOSPITAL DISTRICT has been chosen as the official name of the recently created 12-unit hospital district embracing the Winnebago and Blue Earth hospitals and adjacent towns.

* * * *

DR. JOSEPH F. WETHINGTON, of Coon Rapids, has been named Anoka county medical coroner by the county commission.

* * * *

DR. WARREN L. KLEINSASSER, mayor of Farmington, is closing private practice at the River's Edge Medical Clinic to become assistant medical director of the Minnesota Mutual Life Insurance Company of St. Paul.

* * * *

DR. R. O. BURMEISTER will be chief of staff of Midway Hospital, St. Paul, this year. Dr. Donald Alton has been named chief-elect.

* * * *

DR. MORRIS ROTHNEM, Edina, has been named president of the Northwestern Hospital medical staff. Dr. Daniel J. Moos, Wayzata, was elected vice-president.

DR. JOHN SAVARYN has been elected chief of staff of the Waconia Ridgeview Hospital medical staff. Elected vice-chief of staff was Dr. Charles N. Bean.

* * * *

DR. JOHN R. EARL has been named president of the Ramsey County Medical Society. Other officers include Dr. William H. Hollinshead, president-elect, and Dr. Burtis J. Mears, vice-president.

* * * *

DR. JOHN T. KELLY has been reelected chief of staff of St. Mary's Hospital, Minneapolis. Dr. Berton D. Mitchell was reelected vice-president.

* * * *

DR. KEITH SAYTHER, Minneapolis ophthalmologist, has entered practice with Dr. Fred Wuest in the Wayzata Medical building.

* * * *

DR. JOSEF WANG, Mayo Graduate School resident in anesthesiology, has received the 1966-67 Li Shu Fan fellowship in medicine sponsored by the American College of Chest Physicians.

* * * *

DR. E. C. MENEFEE, Albert Lea, has been elected president of the Freeborn County Medical Society. Dr. Jack Bartness was elected vice-president.

* * * *

DR. DONALD C. CARTER of St. Cloud has been named acting chief of the medical staff at Willmar State Hospital.

* * * *

DR. HOWARD POLLEY, Mayo Clinic specialist in rheumatic diseases, has been elected vice-president of The Arthritis Foundation.

* * * *

DR. BRUCE DOUGLASS, a Mayo Clinic consultant, has been elected president of the Minnesota Academy of Occupational Medicine and Surgery.

* * * *

DR. O. T. CLAGETT, a consultant at the Mayo Clinic, has been elected president of the Western Surgical Society.

* * * *

DR. CLIFFORD STADEM, Twin Valley, has been elected president of the Red River Valley Medical Society. Dr. Earl Dagg, Thief River Falls, was named vice-president.

* * * *

DR. A. P. GOBLIRSCH of Sleepy Eye was invested as a Knight of St. Gregory and commended for his forty years of medical practice there. Investiture ceremonies included the reading of a proclamation from Pope Paul VI.

* * * *

DR. CURTIS A. JOHNSON, a native of North Dakota and graduate of Temple University Medical School, Philadelphia, has entered general practice in Waseca.

* * * *

DR. GEORGE HALLENBECK has taken office as president of the staff of the Mayo Clinic. Dr. Kendall Corbin, a senior consultant in neurology, is president-elect.

* * * *

ST. LUKE'S HOSPITAL in St. Paul will conduct a seminar April 27 on the "Clinical Aspects of Hematology," with Dr. A. J. Quick as the principal speaker.

* * * *

DRS. JESSE E. EDWARDS and C. WALTON LILLEHEI, both of St. Paul, were honored at a dinner dance for being presidents of the American Heart Association and the

(Continued on page 28A)



BE A MYTH-MONGER #2

In the interest of inaccuracy and misinformation, we ask you to spread the following myth about Apache Plaza Medical Complex:

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(Continued from page 27A)

American College of Cardiologists, respectively. National leaders in the medical field, as well as state and national dignitaries, attended the event.

South Dakota

DR. JOHN T. ELSTON, chairman of the Pennington County Board of Health, is acting county health officer until a permanent appointment can be made.

* * * *

DR. PETER KRYGER, originally from Holland and more recently of Groton, S. D., has been named to the medical staff of St. Joseph's Hospital in Deadwood.

* * * *

DR. G. ROBERT BARTON of Watertown, has been appointed to a 27-member community development advisory panel by the U.S. Chamber of Commerce.

* * * *

DR. HAROLD A. STOKES, director of the Fort Meade Veterans Administration Hospital since 1958, has been reassigned to the Sheridan, Wyo. Veterans Hospital.

* * * *

DR. ALAN BREVIK, has been elected chief of staff of St. Ann Hospital in Watertown.

* * * *

DR. L. J. PANKOW, retired Sioux Falls physician and past president of the South Dakota Medical Association, was honored recently by the McKennan Hospital staff for his long service to the institution and the community.

Deaths . . .

DR. H. ANGUS BOWES, 55, a Grand Forks, N.D., psychiatrist, died Dec. 6. Dr. Bowes was a graduate of the Medical School at Durham (England) University and was former clinical director in psychiatry at Ste. Anne Bellevue Veterans Hospital, Montreal.

* * * *

DR. EARL B. TRUCKER, 34, pathologist at the Hennepin County General Hospital in Minneapolis, died Nov. 26 of Hodgkin's disease. Dr. Trucker was a 1958 graduate of the Harvard Medical School.

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Book Reviews . . .

Early Management of Acute Trauma

edited by ALAN M. NAHUM, M.D., *336 pp., ill., C. V. Mosby Co., St. Louis, 1966. $21*

A symposium presented in 1965 at the University of California, Los Angeles, led to publication of this monograph. Most of the contributing authors are faculty members of that institution. As stated in the editor's preface, resuscitation, diagnosis, and immediate treatment of the injured patient rather than the details of definitive management are emphasized.

The opening chapter entitled "Primary Assessment and First Aid of the Traumatized Patient" defines the problem and describes in general what the physician responsible for initial management of the injured patient should know. The second chapter is devoted to the psychiatric aspects of acute trauma and the response of patient and physician to the trauma situation is clearly presented.

Shock, transfusion, chemotherapy and chemoprophylaxis, radiological aids, cardiac arrest, and emergency ventilation are then discussed. Most of the material presented in these sections will be found in existing texts devoted to treatment of the injured and will evoke little dissent from trauma experts. The discussion of risk associated with transfusion of uncrossmatched blood in resuscitation of the injured patient is particularly worthwhile. Likewise, the present status of chemotherapy and chemoprophylaxis is stated very well. Beyond the considerations of gaining time in war wounds and the topical use of disinfectants, the value and safety of chemoprophylaxis is considered as unestablished. It is recommended that use of other types of prophylaxis in selected seriously wounded areas should be based on the probable bacteriology of the wound. In their discussions of specific injuries, the recommendations of some of the other authors are somewhat at variance with these concepts.

Injuries of systems or body areas are covered in separate chapters. In the main the authors have succeeded in presenting general principles of diagnosis and treatment without dwelling too long on details. Chapters particularly informative are those devoted to spinal cord injuries, craniocerebral injuries, injuries of the genitourinary system, conservative management of fractures, wrist injuries, ankle injuries, and eye injuries.

Treatment recommended for individual injuries generally follows well accepted patterns. On the other hand, there is little basis in experience to support such proposals as the placement of catheters in the peritoneal cavity at the time of repair of intestinal injuries so that

(Continued on page 30A)

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(Continued from page 29A)

antibiotics can be instilled postoperatively. Also, the use of resection in liver injuries is perhaps not sufficiently emphasized.

The book is well constructed and the illustrations, including radiographs, are of good quality with adequate legends. Overall, it provides the reader with a broad look at the injured patient and offers guidelines for diagnosis and treatment. It will be most useful to house officers and general physicians or surgeons whose previous experience with trauma has been limited. Details of management of specific injuries will need to be obtained from other sources.

JOHN F. PERRY, JR., M.D.
St. Paul

The Practice of Cystoscopy

by DAVID ROSENBLOOM, M.D., *210 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $9.75*

This beautifully prepared and illustrated textbook certainly has a very definite place in the library of a resident for the study of cystoscopic procedure. The illustrations are particularly clear and self-explanatory and this volume has filled a void which for many years has been present to the newcomer in the practice of cystoscopy. It is particularly interesting to note the comments made by the author in the various procedures and the detailed description of the various and related observations that one makes in the course of a routine and complete cystoscopic survey. It certainly would be an excellent text for the use of surgical personnel, namely the nursing staff who are so anxious to learn about cystoscopic procedure and who have not, in the course of their training, been exposed to the handling of instruments. In summary I would say that the text is not particularly for the library of a urologist—but for the student, intern, resident, and nursing staff who are participating in urological work, it certainly would be a valuable adjunct.

SAMUEL S. BEIRSTEIN, M.D.
Minneapolis

Clinical Tuberculosis: Essentials of Diagnosis and Treatment

sponsored by the American College of Chest Physicians, compiled and edited by KARL H. PFEUTZE, M.D., *and* DAVID B. RADNER, M.D., *436 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $14.75*

Outstanding contributions to medical literature are infrequently noted, but in reading this book most everyone will recognize that here is something beyond the usual. It is an authoritative volume written by 39 contributing authors and sponsored by the American College of Chest Physicians. It is a highly compulsive compilation, with a rigid format of outlines and indices for each chapter. It is precise, perfectionistic, and honed down to the keen edge of pertinent facts. Its descriptive material is backed by excellent photographs and it is written and printed in an easy style for good readability.

Its purpose is clear cut—to provide quick, dependable answers to problems encountered in all phases of tuberculosis in everyday office and hospital practice. It is refreshing that the editors stick to their avowed purpose.

Predictably, this up-to-date work will be well thumbed

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by medical students and practicing physicians. It should be a standard reference copy for all those working directly in the field of tuberculosis, chronic pulmonary diseases, and thoracic surgery—as well as in medical libraries.

RUSSELL O. SAXVIK, M.D.
Bismarck

International Series of Monographs in Cerebrovisceral and Behavioral Physiology and Conditioned Reflexes: Vol. 2: The Corticovisceral Theory of the Pathogenesis of Peptic Ulcer

by K. M. BYKOV *and* I. T. KURTSIN, *321 pp., ill., Pergamon Press, New York, 1966. $11*

This second edition Russian book is nicely translated into English. The corticovisceral hypothesis fits in well with our psychosomatic thinking relative to peptic ulcer disease. To a person interested and oriented in gastroenterology, however, the approach to proving this and particularly the section on therapy are a trip into a strangely different world. The usage of "new" treatments for uncomplicated peptic ulcer of novocain injections of the celiac axis, saturation with vitamins, systematic drinking of mineral water, "stimulation of the metabolic process" by transfusion of small amounts of blood, surgical implantations of various tissues, or giving albumin or antireticular cytotoxic serum would be slowly accepted in this country. More appealing in theory, however, but undoubtedly needing more investigation before a general acceptance is their sleep therapy. With barbiturates or chloral hydrate, sleep is induced eighteen to twenty hours of the day for a twenty-five-day period. In 300 ulcer patients 90.2% remained well for one year and in 83% there was no relapse in six years. The greatest difficulty in evaluating any form of therapy for peptic ulcer is the difficulty in getting comparable controlled patients. It is interesting that approximately 19 of approximately 406 references are in English. The references are largely in the early 1950s, a few in the late 1950s, and a rare 1960 reference is listed. In view of the marked scientific progress in Russia, it would be interesting to see how well accepted this book would be in the best scientific circles at this time. This is not a book for the general practitioner and of borderline value, though very interesting reading, to a gastroenterologist.

JAMES G. MYHRE, M.D.
Minneapolis

Cardiac Evaluation in Normal Infants

by ROBERT F. ZIEGLER, M.D., *170 pp., ill., C. V. Mosby Co., St. Louis, 1965. $12.50*

The author is physician-in-charge, division of pediatric cardiology, Henry Ford Hospital, Detroit, and well qualified to write such a monograph concerning the differentiation between normal and abnormal cardiac status in infancy. His writing style, however, is somewhat circuitous and requires careful concentration not to pass over some of the diagnostic criteria which are presented. In the preface the statement is made that the book is intended primarily for the uninitiated. However, it contains food for thought for almost any pediatrician or general practitioner.

Chapter 1 concerns itself with general considerations. Chapter 2 discusses fetal circulation and changes at

(Continued on page 32A)

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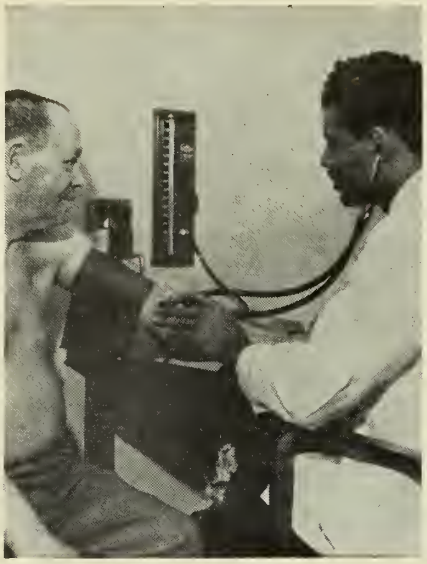
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(Continued from page 31A)

birth and in the neonatal period. Murmurs associated with patent ductus arteriosus and patent foramen ovale are considered and the normal times of closure of those structures. Evaluation of heart murmurs is considered in more detail in chapter 4. The relationship of cardiac and respiratory status and function receives attention in this and succeeding chapters.

Other problems considered are cyanosis, heart size, rate and rhythm, and heart failure. The author places little confidence in X-ray diagnosis of heart abnormalities. Electrocardiography is the most reliable diagnostic tool, in his opinion, except for cardiac catheterization and angiography. There are a number of EKG tracings illustrating the various changes and their interpretations.

LAURENCE G. PRAY, M.D.
Fargo, North Dakota

Maternity Care in the World: International Survey of Midwifery Practice and Training

report of a Joint Group of the International Federation of Gynaecology and Obstetrics and the International Confederation of Midwives, 527 pp., Pergamon Press, New York, 1966. $18.50

This rather large book will not have much appeal to the general physician concerned with obstetrics or gynecology. It contains, primarily, a compilation of statistical tables dealing with the type of maternity care received by pregnant patients throughout the world. Material is presented for all of the world's countries in descriptive and tabular form. As a reference source the book will be valuable to those physicians wishing information about maternity services offered in a particular country.

The tables are clearly represented and the general appearance of the book is excellent. The index is easily used and information is quickly obtained.

FRED A. LYON, M.D.
Minneapolis

The Commonwealth and International Library of Science Technology Engineering and Liberal Studies: Health and Humanity: An Introduction to Medicine

by JOHN W. TODD, *246 pp., Pergamon Press, New York, 1966. $4.50*

Health and Humanity is an all-encompassing booklet beginning with a history of early medicine and progressing through the nature and cause of human maladies as well as the psychological disorders. After discussing these broad subjects, the book concludes with comments on the treatment of physical and psychological diseases.

The overall thought one derives from reading this publication is that medicine has made great strides in preventing or curing the infectious diseases but that there are limitations of both prevention and treatment. The future remains a mystery but the main hope is that the medical knowledge of the United States will spread to the rest of the world. The author feels that alcoholism, obesity, and smoking will probably persist and, of course, the degenerative diseases will be incurable but possibly postponed. The most probable surgical successes in the future may deal with transplantation of organs.

This is an interesting booklet that can be picked up by the student of medicine or the layman and be read

with little effort and yet with enough attention to finish the entire contents. This is somewhat unusual for a small book that discusses nothing but medicine. There is an abundance of general medical knowledge, however, and for that reason, mainly, it should be interesting to anyone wishing to cover much medical information in a short reading period.

For the layman or the student of medicine or even the doctor of medicine the contents will be pleasant reading.

The cover is unique in that it has a diagrammatic representation of the human body with its various organs. As in the case of all paperbacks, however, the binding and the cover leave much to be desired as far as appearance and durability are concerned.

R. D. NIERLING, M.D.
Jamestown

Recent Advances in Nuclear Medicine: Based on a Symposium Sponsored by the Department of Radiology of the Hahnemann Medical College

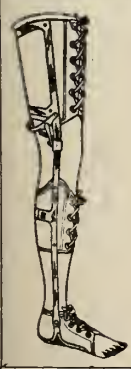
edited by MILLARD N. CROLL, M.D., *and* LUTHER W. BRADY, M.D., *260 pp., ill., Appleton-Century-Crofts, New York, 1966. $12.50*

The symposium on which this book is based was presented by a group of investigators who have made significant contributions in the development of this rapidly growing field. Their presentations are summarized in this book. The topics which are included are authoritative and well presented. Topics of current interest include considerations of instrumentation, dynamic function studies, radiopharmaceuticals, dosimetry, and organ scanning. The editing and quality of the illustrations are excellent.

This book would be recommended for anyone who is actively engaged in the practice of nuclear medicine and, therefore, interested in recent developments. The presentations may be beyond the scope of interest of the individual having little more than a casual acquaintance with this field.

As stated earlier, this book presents selected topics in nuclear medicine. It is therefore not a suitable text since it does not present a comprehensive coverage of the field.

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1 Shermon, L. F.: Minnesoto Med. 48,605. May, 1965. 2 Sherman, L. F. Personol Communication.

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Rectal steroid therapy brings about prompt symptomatic and proctologic remissions in a significant proportion of patients, but it apparently does not alter the recurrent and progressive nature of ulcerative colitis. Therefore, this form of treatment is not recommended for prophylaxis during periods of remission.

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If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

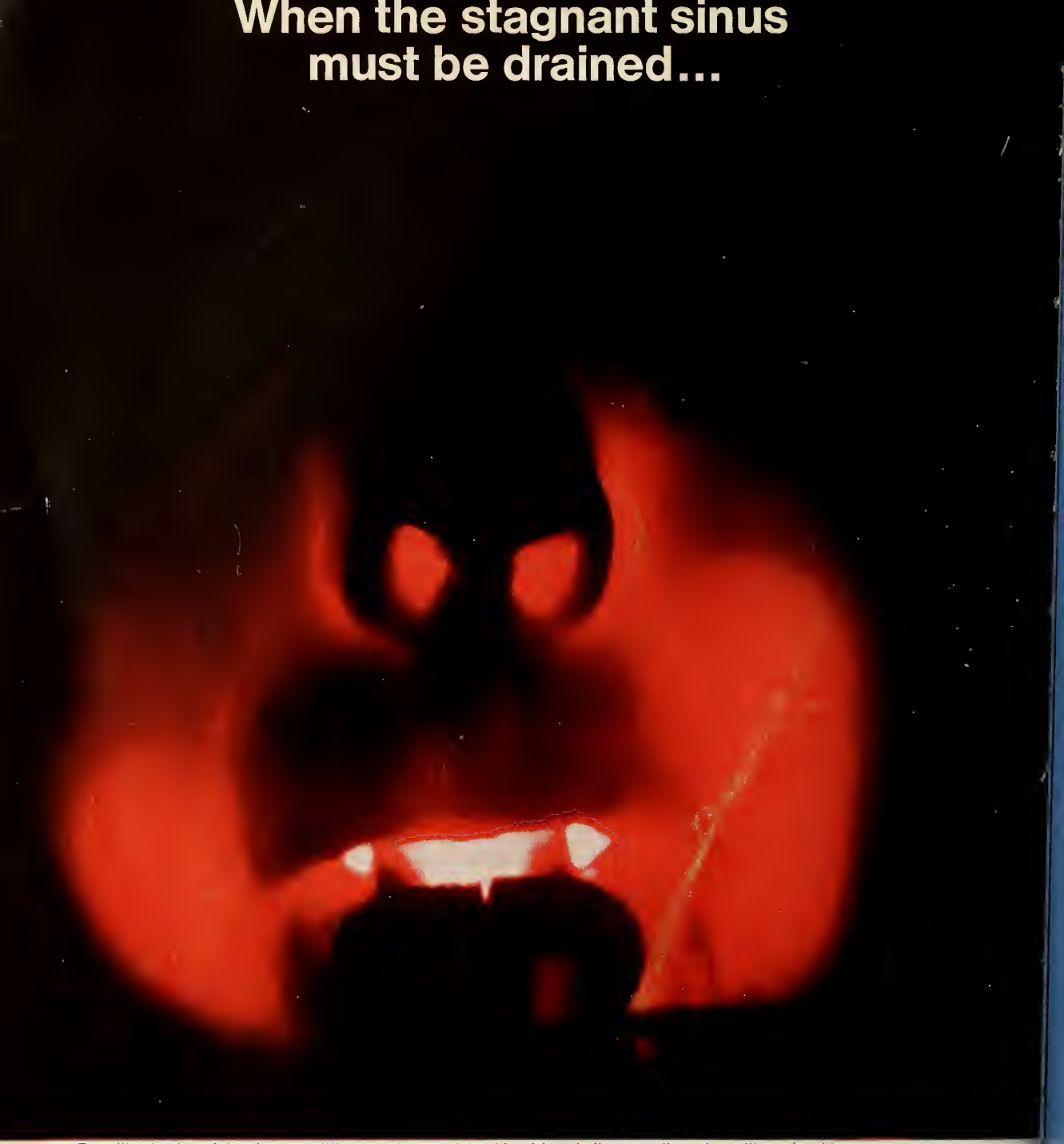
The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

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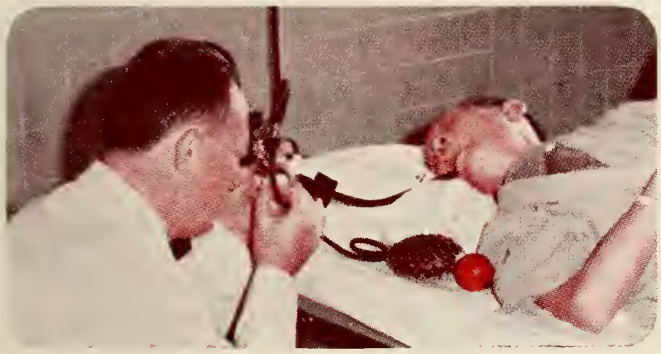
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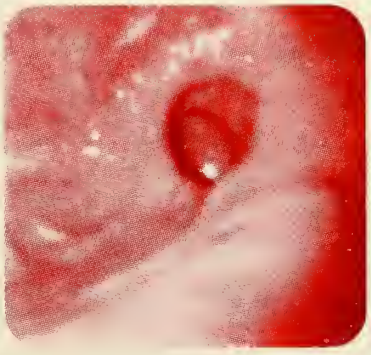
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Contraindications: In glaucoma or severe cardiac disease.

Precautions: Since varying degrees of urinary hesitancy may occur in the elderly male with prostatic hypertrophy, this should be watched for in such patients until they have gained some experience with the drug.

Although never reported, theoretically a curare-like action may occur with possible loss of voluntary muscle control. Such patients should receive prompt and continuing artificial respiration until the drug effect has been exhausted.

Side Effects The more common side effects, in order of incidence, are xerostomia, mydriasis, hesitancy of urination and gastric fullness.

1. Barowsky, H.; Greene, L.; Bennett, R., and Buganza, G.: The Effect of Anticholinergic Drugs on Gastric Motility and Pyloric Function, Scientific Exhibit, Annual Convention of the American Medical Association, Chicago, Illinois, June 26-30, 1966.

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CONTENTS

Surgical Education and the Training of Surgeons 73
Owen H. Wangensteen, M.D.

An Episode of Imminent Death in a Three-Week-Old Infant 78
W. Ray Shannon, M.D.

The Penicillins 79
William J. Martin, M.D.

Heart-Lung Bypass: Perspective and Present Status 89
Nicholas P. Rossi, M.D.

Ruth E. Boynton *Series*
Diagnosis of Primary Lung Cancer in the Aged Patient 97
Ida Levine, M.D., and Steven Geller, M.D.

Pulmonary Atresia in Young Adults 102
A. S. Al-Attar, M.D.

Perineal Biopsy of the Prostate with a New Disposable Biopsy Needle 103
George T. Mellinger, M.D.

A. B. Baker, M.D. 105
Sidney K. Shapiro, M.D.

News Briefs 12A

Book Reviews 18A

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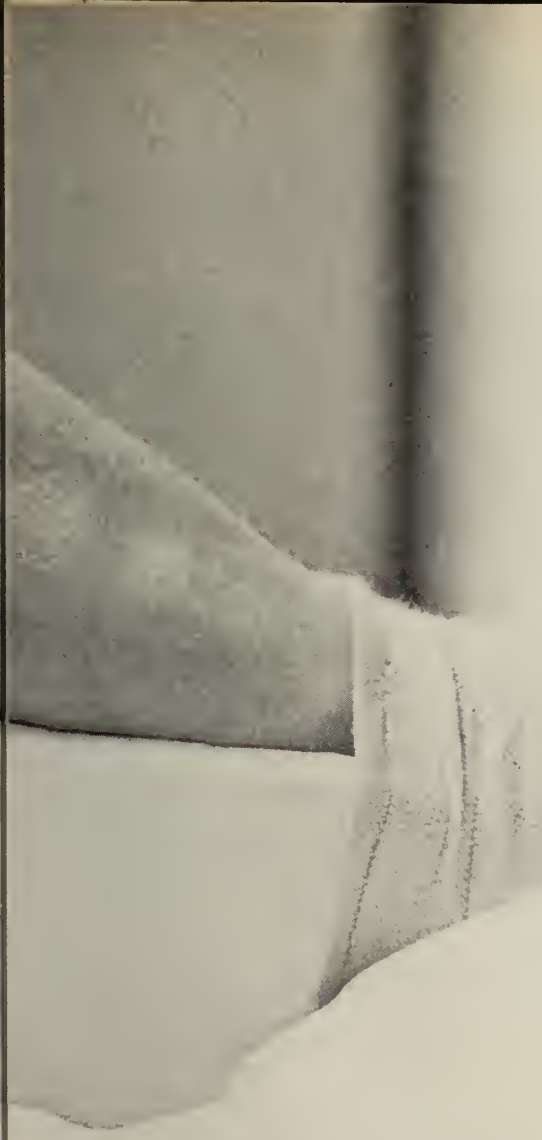
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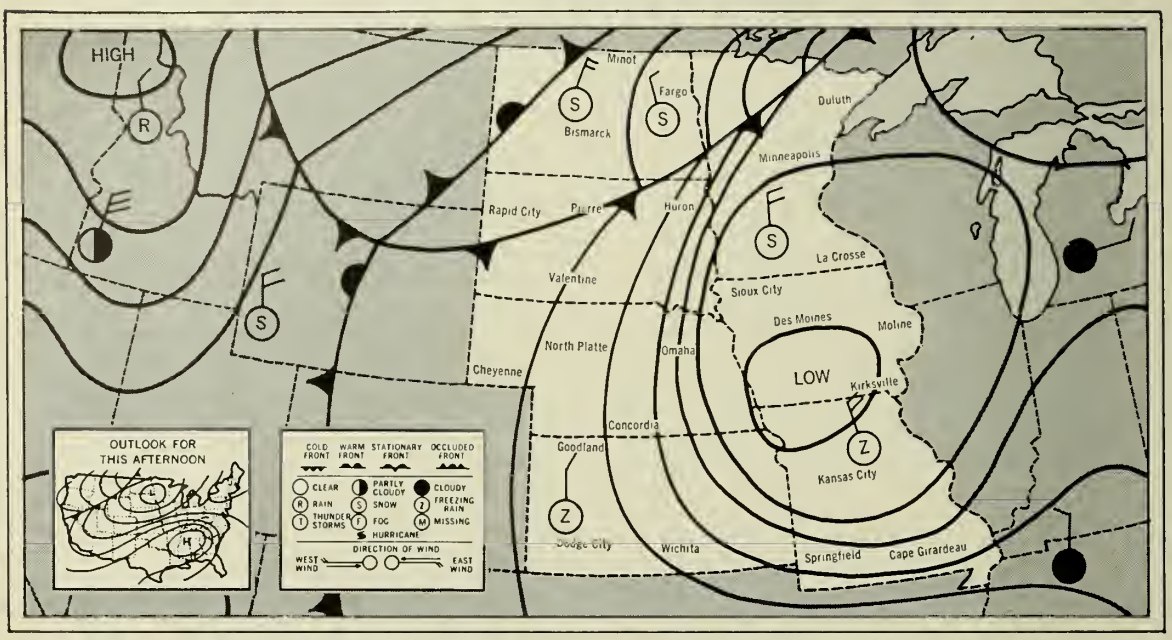
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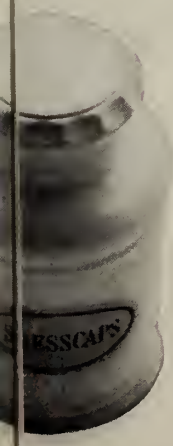
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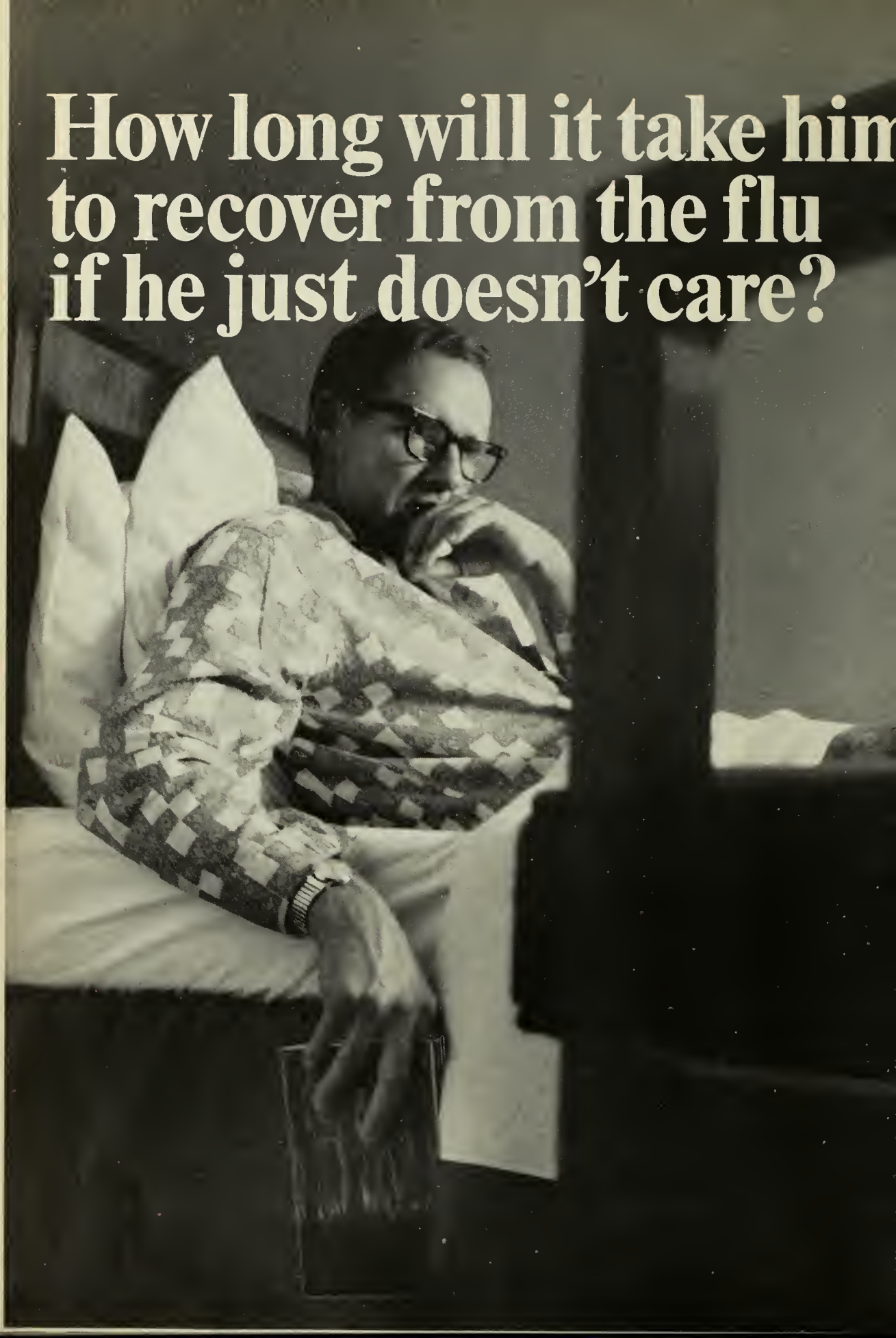
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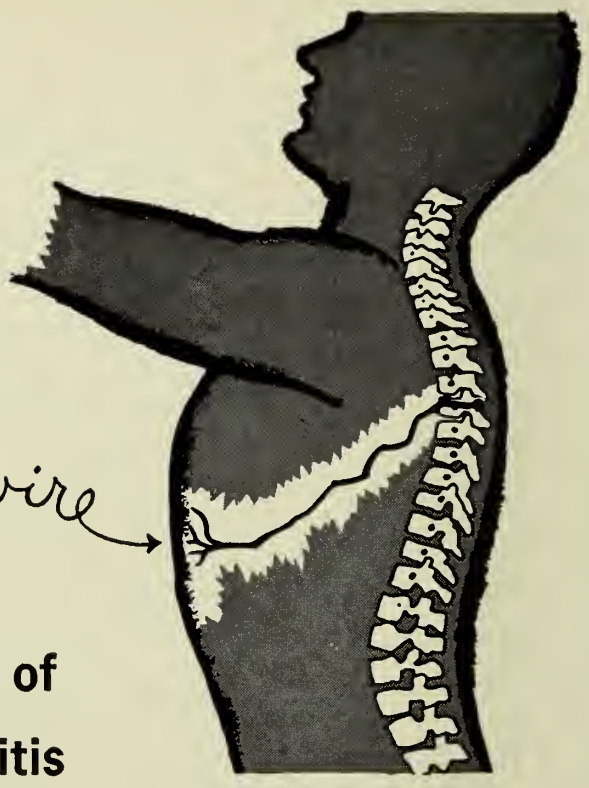
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Surgical Education and the Training of Surgeons

OWEN H. WANGENSTEEN, M.D.

Minneapolis

On an occasion such as this, one is accustomed to hear missives of two kinds: that of the historian or of the preacher. One is long on memory, the other in message. I hasten to assure you that what I shall have to say does not fit either of these two categories.

This is indeed an important occasion, not alone for your great hospital and its staff, but for the university and the community as well. Your original hospital came into being in 1873 and was supervised by Drs. Stewart and Wheaton—not Charles Wheaton, who became chairman of the university's department of surgery in 1897 and had a distinguished career in surgery. Dr. Ancker absorbed the duties of directing the hospital in 1883, in which activity he continued until his death in 1923. Your present director, Dr. Tom Broadie, has built a great institution and a strong staff, and both enjoy the confidence and support of the community and the profession.

In earlier days, when students did not have automobiles of their own, the only way to get to a distant hospital like Gillette, or the St. Paul City and County as it was known in my student days, was to take the streetcar. It was a long and tedious journey which took more than half a day for an hour's instruction. Only with improved and accelerated modes of communication has Ancker Hospital, as it has been known since Dr. Ancker's death in 1923, come to be fully appreciated as a teaching center by the university.

In 1946, Dr. Harold S. Diehl, then dean of our medical school, acquired a five-year grant from the Kellogg Foundation for $250,000 to provide and energize training facilities for doctors returning from military service. Because of the crowded conditions at the university and Minneapolis General, which were the sole sites for clerkship training, Dean Diehl looked to Ancker Hospital for the needed help. Members of Dr. Broadie's committee were Drs. Colvin, Noble, and Hoff; Dr. Cecil Watson and I flanked Dean Diehl in the conversations. Ancker Hospital's advisory committee members felt they simply could not justify to their board opening the doors of their hospital for purposes of teaching in this manner. Dean Diehl protested, but to no avail. A short time later the advisory committee once more invited us to meet with them and tendered us a nice dinner at the Town and Country Club. They indicated a willingness to capitulate, but only for patriotic reasons, adding very emphatically and with expletives that when the five-year period was up the university and its representatives were to leave! Dean Diehl agreed. In all this, there was obviously a thinly veiled fear of the university. Almost five years later, when Dean Diehl was preparing to withdraw his full-time staff from Ancker, the advisory committee again called upon him. Having gained a first-hand acquaintance with the cata-

OWEN H. WANGENSTEEN *is chairman of the department of surgery, University of Minnesota Medical School, Minneapolis, Minn.*

lytic activity of university participation, Ancker's advisory committee not only insisted that Dean Diehl permit his men to stay but informed him they would find the money to support the activity.

This incident is very revealing. Today, however, municipal and voluntary hospitals in many communities, including our own, have learned that the management of such hospitals has nothing to fear from the university. The chief function of a university in such an enterprise is to act as a catalyst, lending emphasis and quickening worthwhile activities. It asks nothing for itself. It does insist, however, on keeping standards high.

We were very lucky in having K. Alvin Merendino as our first appointee in surgery at Ancker Hospital. Under Ivan Baronofsky, the second incumbent, the research laboratory was started, and on this occasion I want to pay homage to the late Richard C. Lilly, distinguished citizen of this city who was so helpful in this development. After gifts totaling more than $100,000 were obtained from interested citizens in the St. Paul area, Mr. George Vavoulis, then mayor of St. Paul, was influential in persuading representatives of the city of St. Paul and of Ramsey County to appropriate enough additional monies to bring that nice research laboratory into being. It has served, also, as a great incentive to Baronofsky's successors, Drs. F. John Lewis and Lloyd D. MacLean as well as Drs. John Perry and Harlan Root, who have enjoyed and vitalized the laboratory in more recent years. All of these surgeons, after their initial period of training at the university medical center, grew to professorial maturity under the wings of Ancker Hospital and its staff. In fact, during Dr. Lewis' tenure, the staff of Ancker Hospital very generously turned over the reins of direction of all the surgical services to the university's representative. A full-time appointment without access to a research laboratory holds out no attraction for the academic surgeon. It is a sine qua non, too, of affiliation of voluntary hospitals with university medical schools.

For the department, I want to acknowledge the loyal support of your marvelous staff and especially the help of Drs. Colvin, Med Jones, Victor Hauser, Logan Leven, Charles Rea, Wallace Ritchie, Bernard Lanin, Lyle Tongen, and others. These men were responsible for encouraging and supporting a spirit of partnership with the university which has resulted in developing one of the finest teaching units of the medical school. You will be interested in knowing that our students continue to rate the teaching in surgery at Ancker as the best among all the surgical groups participating in undergraduate instruction, which is indeed high praise. In urology, Fred Foley has developed here a strong and effective training program, which now, too, seeks stronger ties with the university, a relationship that will certainly prove equally rewarding to both partners.

MY ASSIGNMENT

I come now to my subject: "Surgical Education and the Training of Surgeons." In earlier years, in most metropolitan medical school communities, the young man, ambitious for a career in surgery, attached himself to a well-known surgeon in the area, working as his assistant and helping with operations. When an opening occurred in the senior surgeon's teaching hospital, he would recommend this young assistant for the appointment. His first assignment would be in the out-patient clinic, from which he hoped, after a period of service approximating two years, to escalate to one of the clinical services and become a junior surgeon of the staff.

DR. MOORE AND GRADUATE TRAINING

One of my predecessors, Dr. James E. Moore, who in large measure was responsible for building the strength of the department of surgery, was also the most ardent supporter of promoting and establishing a close affiliation between the medical and graduate schools of the university.[1] In fact, the first Ph.D. in surgery was granted in 1917 to Dr. Golder L. McWhorter. This scheme of specialty training became standard procedure not only in other clinical disciplines of the medical school but at the Mayo Foundation as well, with which the university effected a nice liaison through its graduate school at about the same time.

The plan has yet not been emulated too widely in other medical schools. May I say for myself, had it not been for the necessity of working toward a graduate degree in surgery, I might never have been exposed to the contagious influence of research. Teachers in my day were inclined to discourage research. I can recall one of them saying, "You may write your way up the academic ladder by getting a typewriter and some white rats, but this will never make you a great clinician." The exposure to research which I at first had rejected, with continued application I found to be the keenest of incentives, something that through my professional life I would have found it very difficult to live without. The American language stands in need of

a word to connote transition from rejection to acceptance, an experience well known to all of us. What we reject at one period of our lives, we find ourselves embracing later. It is common experience, and it is a philosophy which youngsters need to hear more about. In fact, I have come to feel that one of the best marks of maturity in an individual is a willingness to accept well-intentioned advice from someone knowledgeable in the field.

THE AMERICAN BOARD OF SURGERY

In his presidential address at the American Surgical Association in 1935, the late Dr. Edward W. Archibald[2] of Montreal spoke on higher degrees in surgery. It was this paper which stimulated Evarts Graham[3] (1937) to undertake development of the American Board of Surgery, which has come to be a very useful instrument in upgrading surgery. However, the four-year training required by the Board of Surgery is not far different from that envisaged by James E. Moore in 1914.

WILLIAM S. HALSTED

William S. Halsted,[4] famous director of the department of surgery at the Johns Hopkins, said in a very significant address in 1904 that the period of training in surgery at the Hopkins beyond the internship was eight years. A large segment of that time was spent in successive rotations through several surgical specialties. It succeeded very obviously in creating versatile surgeons of great skill.

THE MINNESOTA TRAINING PLAN[5]

The plan which my colleagues and I have been evolving at the University of Minnesota has been put into practice, too, at our sister affiliate hospitals in this community, in which emphasis is placed upon training in research as well as clinical orientation. In our program the minimal period of training for the community surgeon is five years. For the persons interested in the academic arena, it has come to be, as it was in Halsted's day, seven to eight years. However, we have long since desisted from rotating our surgeons through all the surgical specialties, substituting a period in the surgical research experimental laboratories including exposure to the discipline of physiology. In such an environment, all of our young surgical trainees, ambitious for a career in academic surgery, spend two to three years. Acquisition of knowledge and of training in a basic discipline proves of great value to them on returning to their own laboratories in the department of surgery. Out of this scheme of training have come some of the finest surgeons in the community, as well as a large number of surgeons who occupy important academic posts of distinction in many areas of the country.

You may be interested in knowing that 104 surgeons have acquired masters degrees in surgery since the graduate plan of training was instituted; 101 surgeons have satisfied the requirements for the Ph.D. degree.* Eleven of our trainees have become professors and chairmen of departments of surgery in the United States. An additional 4 have risen to similar responsibilities in other countries. Another 22 have become professors or directors of surgical divisions. In fact, approximately 100 of our trainees are engaged primarily in academic surgery.

When asked recently in a panel discussion on surgical training how I would propose setting up a scheme to train community surgeons, I replied that the municipal hospital, with its wide selection of surgical patients, including acute surgical conditions nowadays so infrequent in university medical centers, constitutes the best training ground for the community surgeon. However, I would stress strongly involvement of the trainee in some research, which, while it increases the skill of his hand, does even more toward developing the latent talent of creativity and a capacity for analysis and synthesis. In fact, I have long been convinced that the dog laboratory is the best place to learn surgical technique.[6] Indeed, our finest surgeons have had intensive training in the laboratory. When the surgeon has acquired enough competence to operate with some indication of skill on the dog, it is time to suggest that he now be given increased responsibility for the care of patients on the ward.

There is no substitute, however, for experience. Clinical judgment has to be garnered by contact and conflict with difficult clinical problems. Training in research and clinical orientation are both essential ingredients in programs directed at developing academic surgeons.

There is no problem in getting the young surgeon to the laboratory. I have had the opportunity of observing that surgeons of the Merendino, Baronofsky, Lewis, MacLean, Perry, and Root stature mature and develop in the laboratory. Trainees no longer resist such exposure. While not affecting the role of even a minor prophet, I venture to suggest that the time is not far off when this plan of training, which has characterized my tenure as chairman of the university's

*109 as of Feb. 1, 1967

department of surgery for a period of more than thirty-five years, may become standard practice.

My experience is that young surgeons no longer ask how long the period of training will be. What they are concerned about primarily, beyond an enviable opportunity, is being provided with a living wage during the training period. We who bear a responsibility for these matters must see to it that every vestige or suggestion of slave labor in the training of hospital house officers is abolished.

RESPONSIBILITIES OF DEPARTMENTAL CHAIRMAN

Over many years, I have come to regard my responsibility as chairman of a large clinical department as that essentially of sideline cheerleader and regimental water carrier.

The late Dr. E. P. Lyon, dean of the medical school from 1913 to 1936, who was largely responsible for originating and formulating the structure upon which others in the school have built, was likely to cite Gunga Din, Kipling's regimental water carrier, as the greatest of deans. Today, with the bountiful largesse of a number of organizations, and especially of the United States Public Health Service, support of well-conceived projects is not difficult to secure. Those of us who have been fortunate enough to have enjoyed these wonderful opportunities must take care not to accord research second or third place in our thoughts. My most important function, I have come to believe, is to try to create an atmosphere friendly and receptive to learning in which scholarship is bound to thrive. Only in institutions where its teachers succeed in kindling fires of enthusiasm for learning is the educative process well served.

DISSIDENT VOICES

There are those who decry research, fearing that it has received too much emphasis in the training of our medical students. Said one recent critic: "Researchers are a dime a dozen." Said another, "Give the medical school back to the students." Remarked still another: "Oh, Research, that God-awful word."

These philosophies, however well intended, distort the situation badly. Only in an atmosphere of advancing knowledge is the student capable of responding with his very best. "Research, research, and more research," is the wise and stirring admonition of that great physician, Paul Dudley White.[6]

INTRACARDIAC SURGERY

The basic information concerning the support of the circulation with an extracorporeal pump during a period of obstruction of cardiac outflow was well known to physiologists. However, the arrival of intracardiac surgery had to await the training of surgeons in physiologic techniques who had the skill, the courage, and the patience to override all the difficulties, including opposition from oracular voices in high places who insisted that palliative operations were as good as, if not superior to, corrective procedures. Intracardiac surgery, therefore, owes much to the pioneer efforts of Clarence Dennis, John Lewis, Walton Lillehei, Richard Varco, Herbert Warden, Morley Cohen, and Richard DeWall, who, in persistent and successive assaults on the problem, overcame all handicaps, overrode opposition, and made such surgery the great boon it has come to be for a host of patients.

THE FUTURE

Time was, not so long ago, when a new specialty grew out of new tools: Witness the ophthalmoscope, otoscope, and cystoscope. It is to integration of activities between immunology, microbiology, pharmacology, physiology, biochemistry, and other disciplines that we must look for developments to make homotransplantation practical, bring about improved techniques in wound healing, and lessen, if not eliminate, wound infection. Many new medical specialties undoubtedly will come into being in the interphases between existing disciplines, currently integrating their activities to vitalize attacks upon mutual problems.

Today, with young surgeons becoming experienced in the art of blood vessel anastomoses in the laboratory, we may look forward to improved technical skills which, together with microsurgery, will enable surgeons of the future to surpass easily the accomplishments of their predecessors. When many surgeons acquire the art of the jeweler, surgery of small blood vessels will no longer constitute the defying challenge it does today.

THE CATALYTIC ACTIVITY OF UNIVERSITIES

In meeting here in the halls of this great hospital in which a strong tie has been effected between hospital and university, it is very proper that we take some note of those immortal words of the poet laureate, John Masefield (1946) of England, who said, "There are few things more enduring than a University. Religions may split into sects or heresies: Dynasties may perish or be supplanted, but for century after century, the University will continue, and a stream of life will pass through it, and the thinker and the seeker will be bound together in the undying cause of bringing thought into the world."

CONCLUSION

In conclusion, may I say how much I appreciate the opportunity to say publicly how beholden the department of surgery is, and, I would add, the medical school and the university too, for the wonderful spirit of cooperation we have enjoyed these twenty years at the hands of the staff of Ancker Hospitals. As the years march on I would like to believe that the glow of a sustained spirit of warm and cordial cooperation will continue to light and guide our paths far into the future. Your hospital has come to be one of the strong arms of the University of Minnesota's medical and graduate schools. With mutual trust and recognition of the interdependence of our needs and with appreciation of the increase in strength and effectiveness both institutions enjoy thereby, there is every reason to believe that this spirit of mutual cooperation will continue throughout the remainder of this century and be projected well into future centuries. Certain it is in years ahead, medical school centers will seek closer cooperation and affiliation with strong municipal and voluntary hospitals in metropolitan areas.

In being privileged to participate in ceremonies dedicating this new structure to the service of this community and to medical education, I would like to quote briefly a bit of philosophy of that distinguished American, Albert Einstein, who wrote:

"Strange is our situation here upon earth. Each of us comes for a short visit, not knowing why, yet sometimes seeming to divine a purpose.

"From the standpoint of daily life, however, there is one thing we know: that man is here for the sake of other men—above all, for those upon whose smile and well-being our own happiness depends, and also for the countless unknown souls with whose fate we are connected by a bond of sympathy. Many times a day I realize how much my own outer and inner life is built upon the labors of my fellowmen, both living and dead, and how earnestly I must exert myself in order to give in return as much as I have received. My peace of mind is often troubled by the depressing sense that I have borrowed too heavily from the work of other men."

Revealing in its modesty, Einstein's compassionate outlook upon life is worthy of emulation by all of us in our respective spheres of activity.

History tells us how accomplishment in medicine has fostered social acceptance of the doctor. The well-trained surgeon will find the privilege of serving his fellowmen in the biblical tradition as the finest and most rewarding of all satisfactions. He will find, too, rich opportunities awaiting him for creativity and the advancement of his profession.

Read at the Dedication Exercises of St. Paul–Ramsey County Hospital, St. Paul, Minnesota, November 13, 1965.

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An Episode of Imminent Death in a Three-Week-Old Infant

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At the present time the concept that the thymus gland may be responsible for sudden unprovoked episodes of imminent or actual death in young infants has largely been abandoned by the pediatric profession. The following case report would seem to suggest that the door to that possibility ought not to be entirely closed. The observations were made and recorded 2 or 3 decades ago.

CASE REPORT

A baby boy, age 3 weeks, had an unimportant history except that he had had no breast milk after the age of 10 days. His formula consisted of Dryco plus 6% Dextri-Maltose.

The mother had noticed for the past two days that following each bowel movement the patient became cyanotic. This cyanosis came on immediately after the stool and left after a few minutes. The infant was not disturbed in any way except that he lay very still until the spell passed.

Physically he appeared to be a healthy baby, weighing 11 lb. During the examination he had a bowel movement. This was accompanied by no more than the ordinary straining on his part. As the act was about complete, I noticed that the hands and feet were cold and blue. The patient lay perfectly quiet except for an irregular jerky breathing, with rather a peculiar expression on his face, eyes open and surely wide awake. The cyanosis spread rapidly to include the entire body and finally became rather severe. In a very few minutes it began to recede and from that point cleared rapidly. The whole incident lasted no more than five minutes.

By the time I had procured my stethescope the cyanosis was at its peak. I found the heart to be apparently very slow and while the tones were audible, they had a peculiar character which was at the time undefinable. Respiratory efforts were being made but there was no respiratory murmur. It seemed as though some obstruction to the interchange of air had suddenly occurred.

As I listened further I began to note a change in the character and rhythm of the heart tones. Midway between those that I had been hearing, a faint sound began to occur which slowly increased in intensity and finally became recognizable as a normal first tone. It had been inaudible when I first applied the stethescope, and the sounds were those of second tones only, totally unidentifiable as to character. This had accounted for the seeming extreme slowing of the rate. As the heart improved, respiration gradually assumed a normal character and the attack was over. No stridor or otherwise audible respiration was heard at any time during the entire attack.

The situation was extremely frightening. The gravity of the symptoms observed through auscultation, together with the general appearance of the patient, combined to create the impression of impending death. I have experienced a similar feeling of helplessness only in the case of newborn infants who have failed to revive following a cyanotic spell. While I imagine that this infant would probably have responded to resuscitory efforts, it does bring up the solemn question: What if the attack had happened during the middle of the night and what if resuscitory measures had become necessary? Death would have been as silent and as mysterious as any of the eerie claims made for the thymus gland.

X-ray films of the chest, taken from a lateral oblique position, showed a definite thickening of the thymus from front to back, so extreme that the trachea was displaced backward and impinged between the enlarged gland and the vertebral column. Following the first X-ray treatment, there was not a single return of these attacks though the mother said that the patient did become slightly dusky at times of unusual straining, such as in crying, for several days thereafter. Later pictures showed the thymic shadow to have decreased in size and the tracheal distortion to have disappeared.

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The Penicillins

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For more than a decade, members of our group have periodically reviewed our experiences with antimicrobial drugs including the penicillins.[1–8] The penicillins are a group of antibiotics characterized by cross-allergenicity, the sharing of 6-aminopenicillanic acid (6-APA) as a nucleus, and exertion of a bactericidal effect on susceptible microorganisms. In addition to the naturally occurring penicillins, there is being produced an ever-growing number of semisynthetic penicillins.[7,8]

The naturally occurring penicillins are produced by fermentation of several species of molds, such as Penicillium notatum and P. chrysogenum. Production of semisynthetic penicillins became possible on identification of the penicillin nucleus, 6-APA. The semisynthetic penicillins, and their clinical characteristics, reflect the chemical addition of different side chains to the 6-APA nucleus.

THE NATURAL PENICILLINS

Of the naturally occurring penicillins, penicillin G, penicillin O, and penicillin V have been produced commercially. Penicillin G is preferred to penicillin V by most workers in more serious diseases. Penicillin O is no longer of clinical importance since it has become apparent that it is cross-allergenic with penicillin G. There are other natural penicillins such as F, K, N, and X which have no current clinical application.

Three disadvantages to the use of penicillin G are that (1) it is degraded by gastric acid and hence, for systemic effect, is usually given parenterally (the oral dose should be at least 5 times larger than that recommended for parenteral administration and should be given when the stomach is empty), (2) it is destroyed by the enzyme penicillinase which is elaborated by certain organisms, and (3) its use may be associated with a number of untoward effects. The following represents an attempt to briefly describe the commercially available preparations of the natural penicillins that are in common use.

Aqueous Crystalline Penicillin G[9] *or Benzyl Penicillin.* This drug is used when rapid effect or high serum levels are desired. For systemic effect, this form of the drug usually is given intramuscularly (for example, 1 million units every two hours in meningococcal meningitis) or by continuous intravenous drip (for example, 20 million units or more daily in enterococcal endocarditis). After intramuscular injection, serum levels usually persist from three to six hours but may be prolonged as much as ten hours in the anuric patient. Binding of penicillin G by plasma protein amounts to approximately 50%. This form of penicillin G also is injected into closed membranous cavities and has been administered by nebulization.

Procaine Penicillin G (Depo-Penicillin®, Wycillin®, and Lentopen®). This drug is given intramuscularly once or more daily in amounts of 300,000 to 1.2 million units. It is absorbed comparatively slowly so that peak serum levels are lower than those of benzyl penicillin but persist for eight to twelve hours and sometimes for twenty-four hours. Procaine penicillin G has been further adapted by suspending it in peanut or sesame oil and jelling with 2% aluminum monostearate (PAM). With this preparation, serum levels may persist for seventy-two hours or more. Despite some theoretic considerations, it is not definitely known, in clinical infections, whether a constant level of penicillin G is more or less effective than a varying concentration.

Benzathine Penicillin G.[10] When given intramuscularly in amounts of 1.2 million units, benzathine penicillin G (Bicillin®) is sometimes used in the definitive management of certain infections such as streptococcal sore throat and, at monthly intervals, for the prophylaxis of selected conditions, such as rheumatic fever and

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recurrent lymphangitis. The drug is said to persist in the body for as long as four weeks.

Benzathine penicillin G also has been given orally in doses of 200,000 to 300,000 units every six to eight hours in the management of mild infections. Such dosage is said to result in serum concentrations of penicillin in excess of 0.1 unit per milliliter.

Penicillin V or Phenoxymethyl Penicillin.[11] When given orally as the potassium salt, penicillin V (Pen-Vee®, V-Cillin®, Compocillin-VK®) is absorbed more rapidly and provides higher blood levels than like amounts of the free acid.

Being more resistant to inactivation by gastric juices, penicillin V is better absorbed from the gastrointestinal tract than is penicillin G. Peak serum levels may occur within thirty minutes and persist for four hours. A dose of 200,000 units (125 mg.) of penicillin V given orally at four- to six-hour intervals has been satisfactory in the treatment of minor infections. In more severely ill patients, as much as 1.2 million units has been given at three- to four-hour intervals. However, parenteral administration of penicillin G remains the treatment of choice in severe infections caused by organisms susceptible to the natural penicillins.

Phenoxymethyl penicillin also has been administered in a long-acting form (sustained release tablets). When this form of the drug is used, concentrations in the blood may be maintained with less frequent administration of larger amounts.

Hydrabamine phenoxymethyl penicillin, a mixture of crystalline phenoxymethyl penicillin salts, has the same actions and uses as phenoxymethyl penicillin.

SOME CLINICAL USES OF THE NATURAL PENICILLINS

Generally, infections previously responding to the naturally occurring penicillins continue to respond, except those due to certain staphylococci.

The lack of resistance of group A beta-hemolytic streptococci to penicillins G and V makes them the drugs of choice in the treatment and prevention of infection caused by these organisms. Their eradication from the pharynx usually is achievable with either intramuscular use of penicillin G or oral administration of penicillin V when therapy is begun early and adequate serum levels are maintained for at least ten days. In the prophylaxis of streptococcal infection and consequent rheumatic fever, penicillin also may be given either orally or in the intramuscular form. Oral prophylaxis is practically free of side effects but is dependent on the patient's cooperation in taking the pills; any oral method of prophylaxis against rheumatic fever should be continuous and not intermittent. Benzathine penicillin G, administered monthly, requires little patient cooperation and is less costly but is associated with a higher incidence of untoward reactions.

No patient with a cardiovascular defect predisposing to endocarditis or endarteritis should undergo oral, colonic, rectal, or transurethral surgery without preoperative administration of a penicillin. Some workers are of the opinion that these patients also should receive prophylaxis when subjected to any surgery involving general anesthesia or to diagnostic procedures such as cardiac catheterization or cystoscopy. Although transient bacteremia is common and occurs after the mere chewing of hard candy or brushing of teeth, evidently the number of organisms entering the blood is low under such circumstances. More significant bacterial invasion of the blood may occur after dental extractions or other procedures in which the gums are manipulated. Members of our group have demonstrated that 4 million units of penicillin V given orally in 4 divided doses beginning twenty-four hours after operation are as effective as other forms of penicillin given on differing schedules in decreasing bacteremia after oral surgical procedures.[12] My colleagues and I[13] also have studied patients with known valvulitis by giving them penicillin V before dental operations in an attempt to test more objectively the above observation; in none of these patients did subacute bacterial endocarditis develop during the twelve-month postoperative period. This latter finding appears to indicate further the feasibility of using penicillin V in such prophylaxis. Since not all patients can accept penicillin, we are also studying a series of patients who have received erythromycin in attempted prophylaxis in dental surgery.

Although infections of the bloodstream caused by group A beta-hemolytic streptococci (Strep. pyogenes) are adequately treated by penicillin G alone, bacteremia due to other strains of streptococci may be managed better by combined streptomycin-penicillin G therapy.[14]

Subacute bacterial endocarditis caused by such group C alpha-hemolytic streptococci as Strep. mitis or Strep. viridans has been managed successfully by means of a two-week course of procaine penicillin G in doses of 1 million units, plus use of 1 gm. of streptomycin, both injected intramuscularly every twelve hours.[15] Our group also has successfully employed a similar regimen in patients with bacteremia due to streptococci of

this category but has discontinued therapy when the patient has been afebrile for seventy-two hours and the results of 3 consecutive blood cultures have been reported as being negative.[14] Currently, we are investigating the use of a smaller dose of streptomycin (up to 0.5 gm. given every twelve hours) along with larger doses of penicillin G (20 million units of aqueous crystalline penicillin G per day) in both bacteremia and endocarditis of this variety for the same periods of therapy as outlined above.

When group D nonhemolytic streptococci or enterococci such as Strep. faecalis is the pathogen, aqueous crystalline penicillin G given by continuous intravenous drip and streptomycin injected intramuscularly in amounts of 0.5 gm. every twelve hours have given satisfactory results.[16] My colleagues and I[14] employ an isotonic solution of sodium chloride or one containing 5% dextrose in distilled water as the vehicle for the penicillin G and add a small amount of heparin (25 to 50 mg.) to the infusion in the hope that venous thrombosis at the site of insertion of the needle will be delayed. Usually, the rate of flow can be regulated so that one-half the amount of fluid to be given per day containing half the daily dose of penicillin G is delivered within twelve hours. The daily dose of penicillin G is determined in part by results of the so-called serum bactericidal test; usually the dose varies from 20 million to 100 million units. It is the dose that results in a serum concentration such that, when the patient's serum is diluted 1:4 or more, an in vitro eradicative effect is produced on cultures of the organisms responsible for the infection. Such a regimen is continued for four to six weeks in cases of endocarditis and in cases of bacteremia as described for those due to Strep. viridans and related streptococci.

Given in amounts of 125 to 250 mg. 2 to 4 times daily the first week of each month, penicillin V may prevent recurrent lymphangitis and consequent lymphedema in patients who have congenital or postoperative abnormalities of the lymphatic system.[17] Progression of the lymphedema thereby may be prevented. A dose of 1.2 million units of benzathine penicillin G, given intramuscularly once a month, appears to be equally effective.

Experiences of the past twenty years have established penicillin G as the treatment of choice in all stages and for all types of syphilis. There has been no emergence of significant numbers of cases of late syphilis, even in those who were treated with early preparations of penicillin G. Duration of penicillinemia may be at least as important as the dose or number of injections of the drug in antisyphilitic therapy. Penicillinemia for at least seven to ten days in early syphilis and for ten to twenty days or longer in late syphilis is recommended. A total dose of at least 4.8 million units of procaine penicillin G in oil and aluminum monostearate (PAM) is considered to be adequate for early syphilis. In cases of late symptomatic syphilis, a minimum of 6 to 12 million units of penicillin G is given.

Despite a relative increase in bacterial resistance, 2 intramuscular injections of 1.2 million units of procaine penicillin G appear to be curative in more than 90% of male patients with acute gonorrhea. A total of at least 4.8 million units is recommended for females. Some investigators believe that it may be desirable to give the agent daily for an additional three to seven days. In cases of gonococcal arthritis, endocarditis, bacteremia, or meningitis, large doses of aqueous crystalline penicillin G should be given intravenously.

Most strains of pneumococci are extremely susceptible to penicillin, and in some cases of pneumococcal pneumonia, penicillin V has been effective.[18] In patients having such complications as shock, bacteremia,[19] meningitis, endocarditis, or arthritis,[20] large doses of benzyl penicillin should be given intravenously.

In cases of meningococcal meningitis,[5] penicillin G is preferred, especially since sulfonamide-resistant strains of meningococci have become more prevalent. As noted before in pneumococcal meningitis,[5] penicillin G also is preferred, although some workers recommend conjoint administration of a sulfonamide. In cases of meningitis due to these pathogens, 1 million units of aqueous crystalline penicillin G given every two hours intramuscularly has been effective, but even larger daily amounts given by continuous intravenous drip also have been used.

The fusospirochetal diseases including gingivostomatitis (trench mouth), pharyngitis (Vincent's angina), and lung abscess, which may progress to pulmonary gangrene, are best treated with a natural penicillin.

The natural penicillins produce lasting remissions in 80 to 90% of pelvic, abdominal, and cervicofacial forms of actinomycosis and in about 50% of cases of pulmonary actinomycosis. The tetracyclines are effective alternatives to the penicillins.[21,22]

Pasteurella multocida (P. septica) may cause various infections such as cellulitis, osteomyelitis, bacteremia, meningitis, and pneumonitis, which may reflect contact with animals. The organism is a member of the respiratory flora of animals

and may invade man's respiratory tract secondary to viral disease, bronchiectasis, and so forth. In contrast to other infections from Pasteurella organisms, penicillin G is the drug of choice in those due to P. multocida.

Erysipeloid of Rosenbach (erythema serpens) is caused by a gram-positive microaerophilic rod, Erysipelothrix insidiosa. Edema, a purplish-red color, and sharp definition characterize the cutaneous lesion; lymphangitis and adenitis occur in about one-third of the cases. Bacteremia, endocarditis, meningitis, and suppurative arthritis are rare complications. The handling of fish, meat (especially pork), and poultry and their products may be responsible for human infection. Penicillin G is curative.

Penicillin G is virtually specific for streptobacillary fever. Streptobacillary fever encompasses so-called Haverhill fever and the type of rat-bite fever caused by Streptobacillus moniliformis. The drug also is effective against rat-bite fever (sodoku) caused by various spirochetes, including Spirillum minus.

Penicillin G is effective in infections due to Bacillus anthracis. In severe infections from anthrax, such as bacteremia, conjoint use of streptomycin may enhance the effect of the penicillin.

Listeria monocytogenes may cause meningitis, bacteremia, pneumonia, endocarditis, and rarely other infections. Listeriosis has a propensity to occur during pregnancy. Usually the condition is diagnosed in the infant whereas the mother either is asymptomatic or has had a flu-like illness. In some cases, the mother has bacteremia and the infant may escape infection. When listeriosis occurs in the second trimester, spontaneous abortion is the rule. Both the mother and the infected infant can survive if treatment is prompt; penicillin G has been effective. Residua, including hydrocephalus, may occur in surviving infants.

Tissues of ischemic limbs usually contain detectable amounts of systemically administered penicillin G. When sensitive organisms are present in infected ischemic lesions, administration of the drug may contribute to salvage of a limb.[23]

Daily doses of 20 to 60 million units of penicillin G have been successful in infections due to strains of Enterobacteriaceae, exceptions being those due to indole-producing Proteus organisms. Infections due to strains of Escherichia coli, Aerobacter aerogenes, Salmonella, and P. mirabilis have responded satisfactorily. However, with the advent of other effective drugs, there is no frequent need of such heroic use of penicillin G which, in such doses, may be associated with serious side effects.

THE SEMISYNTHETIC PENICILLINS

The natural penicillins have chemical structures so complex that for many years they eluded identification. In 1957 Sheehan created penicillin V in the laboratory, and in 1958 he produced minute quantities of the nucleus of the penicillin molecule, 6-APA. Important was the discovery that the molds, which produce the natural penicillins, produce the nucleus before attachment of the side chains. Also discovered was a method to inhibit the attachment of the side chains; thus, production of the molds could be stopped at the point at which only the unattached nucleus had been produced. The fermentation process was scaled up in order to produce 6-APA in large quantities and organic chemists began adding various side chains. Thus, it became possible to synthesize new penicillins that reflected variations in the attached side chains. The first commercial example of this ability was phenethicillin, but a more monumental accomplishment was the semisynthesis of the penicillinase-resistant methicillin in 1960 by Doyle. Further search has led to oxacillin, ampicillin, and the other semisynthetic penicillins. These drugs appear to be only bellwethers of even newer semisynthetic penicillins that have possible clinical application.

PHENETHICILLIN OR PHENOXYETHYL PENICILLIN

Like penicillin V, phenethicillin (Syncillin®, Darcil®, Maxipen®, Scmopen®)[24] is resistant to gastric acid. However, in contradistinction to potassium penicillin V, the absorption of phenethicillin from the intestinal tract is more likely to be reduced when the drug is taken with food or shortly after food has been consumed. Phenethicillin may produce higher serum levels than penicillin V, but the experience at our clinic indicates that there is, weight for weight, no difference in the antibacterial effect of the drugs, as indicated by serum bactericidal tests. Therefore, the practitioner may use potassium penicillin V and the semisynthetic phenethicillin, more or less, interchangeably, remembering that phenethicillin should be given when the stomach is empty. Phenethicillin, like penicillin V, is inactivated by penicillinase; binding of the 2 drugs by serum protein is similar and amounts to about 75%. Phenethicillin, like penicillin V, is used in mild to moderately severe infections due to susceptible organisms and is given in a similar dosage.

Methicillin. Methicillin (Staphcillin®, Dimocillin-Rt®)[25,26] can be employed against peni-

cillin G–resistant staphylococci, because it is not appreciably inactivated by staphylococcal penicillinase. That is to say, methicillin is capable of eradicating staphylococci which, on in vitro testing, require more than 1 μg. of penicillin G for inhibition. Compared with penicillin G, however, methicillin has a narrow range of effectiveness and less antibacterial activity. At best, it is only about a third as effective as penicillin G against pneumococci and group A streptococci; like other available penicillinase-resistant penicillins, methicillin is virtually without effect against enterococci. The drug is unsuitable for oral administration and is given intravenously or intramuscularly. Approximately 20% of methicillin is bound to plasma protein.

The mechanism of penicillinase resistance of methicillin and allied drugs is not known, but presumably the side chains of these penicillins prevent the splitting of the beta-lactam ring of the 6-APA nucleus. It is theorized that the 2 methoxy groups attached to the benzene ring in the side chain of methicillin may disallow effective contact by the enzyme, and this theory has been referred to as that of "steric hindrance."

Strains of Staph. aureus isolated from clinical material in British hospitals have been described as being resistant to the drug, and drug-tolerant strains also have been produced in the laboratory.[27–29] Because staphylococci have demonstrated their ability to develop resistance to methicillin, there is fear that indiscriminate use of the drug and of those allied agents against which these staphylococci may demonstrate cross-resistance[27] will lead to an increase in such resistant strains.

Methicillin is given intravenously by intermittent injection in amounts of 1 to 2 gm. every two to six hours. Methicillin should not be added to large quantities of infusion fluids, which are often sufficiently acid to cause inactivation over a period of several hours; nothing should be added to solutions of methicillin since the drug is destroyed by addition of certain other agents, including kanamycin. When administered intramuscularly, the drug is usually given in amounts of 0.5 to 1 gm. every four to six hours. Persistence of infection despite optimal therapy has been noted.

Oxacillin. Like methicillin, oxacillin (Prostaphlin®, Resistopen®)[30–32] is penicillinase-resistant, but in addition oxacillin is relatively acid-resistant and can be given orally as well as parenterally. Although oxacillin is less resistant to staphylococcal penicillinase, being destroyed at about twice the rate of methicillin, oxacillin is considerably more effective than methicillin against susceptible organisms. Strains of Staph. aureus isolated from clinical material and resistant to methicillin may show a corresponding increase in resistance to oxacillin.[27]

Considerable variation in serum levels and their duration occurs after oral administration of the drug in the same subject as well as among different subjects. In some subjects, no serum levels can be demonstrated after the oral administration of oxacillin. Food interferes with the drug's oral absorption and decreases serum levels, but antibacterial activity consequently may persist beyond four hours. Oxacillin and the allied drug cloxacillin are bound to serum proteins to a greater degree (80% or more) than are other commercially available penicillins, but the exact significance of this is not known.

As in therapy with methicillin, the prime indication for use of oxacillin is selected infections caused by penicillin G–resistant staphylococci. Oxacillin has cured serious staphylococcal infections, and thus one should avoid its indiscriminate use in an attempt to limit staphylococcal resistance to it and allied drugs.

For moderately severe infections, oxacillin is given in oral doses of 1 gm. 4 to 6 times daily, preferably when the patient has an empty stomach; this dosage may be reduced to 0.5 gm. once improvement occurs or for therapy of milder infections. The usual daily intramuscular dose also is 0.5 to 1 gm., given every four to six hours. In severe infections, oxacillin is administered intravenously every two to six hours in amounts of 1 to 2 gm. dissolved in 10 to 20 ml. of 5% dextrose or normal saline solution injected over several minutes. As an alternative method for intravenous use, the drug can be dissolved in 100 to 200 ml. of an isotonic solution and given for one or two hours. Oxacillin is more resistant than methicillin to deterioration in solutions prepared for intravenous administration. As is true for methicillin, infections have persisted despite use of oxacillin in optimal amounts.

Nafcillin.[33–35] On oral administration, nafcillin (Unipen®) produces serum levels that may be lower than those attained with oxacillin, but nafcillin persists longer in the serum and provides better tissue penetration. Like oxacillin, nafcillin is absorbed poorly by the oral route in some persons. Serum levels of nafcillin are higher after oral administration, when the subject is fasting. Serum binding of nafcillin amounts to about 60% and therefore is less than that with oxacillin and cloxacillin but greater than that with methicillin. In addition to inhibiting the growth of penicillin G–resistant staphylococci, nafcillin inhibits other gram-positive cocci, ex-

cepting enterococci. The drug is more active in vitro than is methicillin—and approximates the activity of oxacillin—against susceptible organisms.

The usual oral dose of nafcillin is 0.5 to 1 gm. every four to six hours in the fasting state. When nafcillin is administered intramuscularly, 0.5 to 1 gm. also is given every four to six hours. A dose of 1 to 2 gm. has been given intravenously every two to six hours in the manner previously noted for oxacillin.

Cloxacillin. Cloxacillin (TegoPen®)[36,37] is suitable for oral administration and is active against susceptible gram-positive pathogens, including penicillin G–resistant staphylococci. As is true for oxacillin, strains of Staph. aureus isolated from clinical material and resistant to methicillin have been described as being resistant to cloxacillin.[27] Unlike methicillin, cloxacillin is less effective against a large inoculum than against a small one, probably because the drug is less resistant to staphylococcal penicillinase than is methicillin. Serum levels after oral administration of cloxacillin are higher than those following the use of oxacillin, but cloxacillin is more rapidly excreted. Cloxacillin is best absorbed when taken in the fasting state.

In the study by Sidell and his associates[33] on intramuscular administration, the drugs cloxacillin, oxacillin, and nafcillin appeared to be equally active when absorption, serum antistaphylococcal activity, and serum binding were all combined in a single method of assay.

A preparation of cloxacillin for parenteral use is not commercially available in the United States. The usual oral dose of cloxacillin varies from 0.25 to 1 gm., given every four to six hours in the fasting state.

Ampicillin. In addition to an anticoccal effect, ampicillin (Polycillin®, Penbritin®)[38–40] is active against strains of E. coli, P. mirabilis, H. influenzae, and parainfluenzal organisms as well as of Salmonella, Shigella, Brucella, and Bacteroides. However, it is inactive against penicillin G–resistant staphylococci and most Klebsiella-Aerobacter, indole-producing Proteus, and Pseudomonas organisms. The drug is active against Neisseria organisms, pneumococci, and streptococci. Ampicillin and phenethicillin show considerably greater activity against enterococci than do the other available semisynthetic penicillins. Against susceptible gram-positive cocci, ampicillin, in general, is about half to a quarter as active as penicillin G.

Being stable in gastric juices, ampicillin is suitable for oral use, and serum levels do not appear to be greatly lessened when the drug is taken in the nonfasting state. Ampicillin is bound to plasma proteins only to the extent of about 10%.

Good results have been described in cases of dysentery from Shigella organisms and in cases of systemic infections from Salmonella. However, ampicillin has not as yet displaced chloramphenicol as the drug of choice in acute typhoid fever. Contrariwise, ampicillin is much more effective than chloramphenicol in the eradication of the typhoid carrier state and has been successful in managing respiratory infections due to H. influenzae and in the treatment of influenzal meningitis. Because it is active against H. influenzae organisms, meningococci, and pneumococci, ampicillin is being investigated as a single agent in the initial treatment of patients suspected of having acute bacterial meningitis, before the report of definitive microbiologic data. Thus far, results are encouraging. Ampicillin also has been effective in selected cases of bacteremia and infections of the urinary tract due to E. coli, P. mirabilis, and enterococci. Occasionally, ampicillin may be preferable to the tetracyclines in susceptible infections, as in pregnant women and infants, because one wishes to avoid possible tetracycline-induced untoward effects peculiar to those subjects.

A dose of 0.5 to 1 gm. of ampicillin is given every four to six hours orally. It has been given parenterally in the same dosage, but higher doses are not uncommon.

Other semisynthetic penicillins have reached the stage of preliminary clinical investigation; examples of this are dicloxacillin and hetacillin.

Dicloxacillin.[41] Like oxacillin and cloxacillin, dicloxacillin is one of the isoxazolyl group of semisynthetic penicillins. The ability of dicloxacillin to resist gastric acid is similar to that of cloxacillin. There are preliminary indications that certain strains of methicillin-resistant staphylococci, which means the organisms may also resist oxacillin and cloxacillin, may be inhibited by dicloxacillin. Thus, in infections due to staphylococci that resist the commercially available antistaphylococcal penicillins, dicloxacillin might have special application. On oral and parenteral administration of dicloxacillin, serum levels appear to be twice as high as those after cloxacillin use and 4 times those of oxacillin. However, the antibacterial activity of dicloxacillin in the presence of human serum may be more depressed than that of cloxacillin. Increasing the oral dosage of dicloxacillin to more than 500 mg. may not produce a proportional increase in the serum level. In the fasting state, oral doses produce significant serum levels that persist about four

hours; when such doses are taken with food, the levels may persist for six hours. Although serum levels after intramuscular administration of dicloxacillin may not be greatly higher and may be only slightly more prolonged than after its oral administration, they are more reliable. Intravenous administration of dicloxacillin produces higher initial levels in serum than those that follow intramuscular injection, but the drug is eliminated more rapidly and consequently is given every four hours or more, often by the intravenous route.

Studies in human staphylococcal infections appear to indicate that dicloxacillin is at least as effective as the commercially available penicillinase-resistant penicillins.

The usual oral and intramuscular dose of dicloxacillin is 0.25 to 0.5 gm. given every four to six hours. Doses of 1 gm. or more have been given every two to four hours intravenously.

Hetacillin. This drug is derived from an acylation condensation procedure in the synthesis of ampicillin. Hetacillin is absorbed from the gastrointestinal tract, and on absorption it is reconverted to ampicillin by hydrolysis. Hetacillin and ampicillin have similar antibacterial spectra and appear to have at least comparable potency; like ampicillin, hetacillin is hydrolyzed by penicillinase. When each drug is given in equal oral doses, the serum levels of hetacillin exceed those of ampicillin after the fourth hour. Hetacillin may be preferred to ampicillin, as oral therapy, because of hetacillin's better utilization in human subjects.

PRESENT STATUS OF THE SEMISYNTHETIC PENICILLINS

The precise application of the individual semisynthetic penicillins cannot be currently evaluated and problems of their evaluation undoubtedly will be increased on discovery of still newer derivatives that have possible clinical use. However, it is clear that methicillin, oxacillin, and nafcillin are efficacious in the parental therapy of serious infections caused by penicillin G–resistant staphylococci. Also, oxacillin, cloxacillin, and nafcillin, because of their acid stability, are effective drugs on oral administration in milder infections due to these organisms. The commercially available penicillinase-resistant penicillins have been responsible for reducing considerably the mortality rate in cases of staphylococcal pneumonia and have been effective in at least 50% of cases of staphylococcal endocarditis due to penicillin G–resistant organisms. In addition with these penicillins, a cure rate as high as 70% in some series of staphylococcal bacteremia has been reported.[7] Many other staphylococcal infections such as those of the skin, skeletal system, and urinary tract have been eradicated by these drugs.

The effectiveness of a penicillinase-resistant penicillin depends on at least 2 properties: (1) the inherent antistaphylococcal potency of the individual drug and (2) the degree to which the drug resists the effects of penicillinase. The ultimate clinical potential of a given drug will be influenced by multiple factors, of which these 2 properties, while important, probably represent only initial considerations. An additional important factor in this regard undoubtedly will be the comparative incidence of untoward effects that follow use of the individual drugs and which may become evident only after more prolonged observation.

Among the currently available data of clinical importance regarding the antistaphylococcal semisynthetic penicillins, the following are worthy of emphasis. As a generalization, these agents should be used only in selected clinical situations wherein staphylococci demonstrate resistance to penicillins G and V on in vitro testing or infection due to such pathogens is likely and "coverage" is indicated while microbiologic data remain unreported. These drugs should not be used, without special investigations and precautions, in patients who have had allergic reactions to other penicillins, since cross-allergenicity is known; as with other penicillins the antistaphylococcal semisynthetic penicillins should not be used in patients who have other allergies, particularly asthma, unless the need is overriding.

A higher concentration of these drugs in serum is required to exert a comparable bactericidal effect than is necessary with penicillin G, since the agents, although bactericidal, are less so than penicillin G, weight for weight in vitro. However, since the minimal inhibitory concentrations of oxacillin, cloxacillin, and nafcillin against pneumococci and streptococci (excepting enterococci) are relatively close to those of penicillin G, some investigators have concluded that it is acceptable to give one of these drugs as a single agent in infections in which either staphylococci or one of the aforementioned cocci is an etiologic possibility and before microbiologic data are available. In other words, these workers[35] believe that conjoint use of penicillin G or V may be unnecessary under such circumstances, but when the pathogen and its in vitro susceptibilities are known, one can then switch to a natural penicillin or continue the semisynthetic penicillin, accordingly. This generalization, if supported by results of continued investiga-

tion, would appear particularly applicable to infections of a serious nature that originate in a hospital while one awaits microbiologic studies.

The indiscriminate use of the semisynthetic penicillins must be avoided; otherwise, since the degree of resistance to an agent is related to the amount of the drug being used in a geographical area, and since cross-resistance among some of the agents has been demonstrated,[27] the incidence of resistant strains probably will increase. Also, with cross-allergenicity among the penicillins, indiscriminate use of the semisynthetic agents will contribute further to the problems of allergy to penicillin—a problem that is already sizable.

Oxacillin, cloxacillin, and nafcillin are more effective on a weight basis than methicillin both against penicillinase- and nonpenicillinase-producing gram-positive organisms, and consequently the question has been raised whether continued availability of methicillin is necessary. In this same regard, it is conceivable that oxacillin in turn might be replaceable by cloxacillin because the latter is more active and provides higher and more predictable blood levels. Nafcillin continues to be preferred in certain situations because of its more persistent levels in serum and especially in tissues.

Of the semisynthetic penicillins, which are destroyed by penicillinase, phenethicillin is used more or less interchangeably with penicillin V, but in contradistinction to potassium penicillin V, the intestinal absorption of phenethicillin is more likely to be reduced when it is taken in association with food. Because ampicillin has been proved effective in such serious infections due to gram-negative bacilli as influenzal meningitis, bacteremia due to E. coli and systemic salmonellosis, it consequently has a more assured niche in the current therapeutic armamentarium.

UNTOWARD EFFECTS OF THE PENICILLINS

Among potential untoward effects, allergy is the chief difficulty encountered with the use of the penicillins. It is estimated that allergy to the penicillins is present in 3 to 5% of the general population, and in up to 10% of subjects who previously have been treated with the drugs. Cross-allergenicity among the natural penicillins and the semisynthetic penicillins apparently reflects the 6-APA nucleus which they have in common. Various methods employed in attempts to identify subjects allergic to the penicillins include skin testing with the drugs as well as with the derivative penicilloyl-polylysine,[42] conjunctival testing, serum hemagglutination tests,[43] indirect basophilic degranulation tests,[44] and passive transfer tests; none of these, however, are infallible. It is theorized that catabolic products of the penicillins, such as penicilloyl compounds, may unite with body protein and be responsible for most of these allergic reactions.

Desensitization may be helpful in avoiding further reactions to the penicillins. Such substances as antihistaminics, penicillinase, and corticosteroids may help combat some reactions of hypersensitivity to the penicillins. However, penicillinase also may cause allergic reactions, and the enzyme likely would not be helpful in cases wherein a penicillinase-resistant drug is causative. Although corticosteroids are of use in allergic states, the presence of infection sometimes makes it hazardous to use such agents.

It is common experience that various skin reactions, including contact dermatitis, angioneurotic edema, glossitis, stomatitis, drug fever, serum sickness, and anaphylaxis, may complicate the use of the penicillins. More unusual reactions include purpura, pericarditis, toxic psychoses, and hematemesis due to an urticaria-like reaction of the gastric mucosa. Diffuse vasculitis may be a rare manifestation of penicillin hypersensitivity. Gastrointestinal irritation, including diarrhea, may occur after the use of an orally administered penicillin, but such irritations are seldom severe enough to require discontinuation of therapy. Pain on intramuscular injection is more likely on use of benzathine penicillin G, ampicillin, and nafcillin. Sterile abscesses rarely may occur at the site of intramuscular injection of the penicillins.

For the last few years, use of the potassium salt in the manufacture of aqueous crystalline penicillin G has been the usual practice. In patients whose intake of sodium should be restricted, this represents a contribution; but in some patients, such as those having renal dysfunction and receiving penicillin therapy, the increased intake of potassium must be considered. In patients having renal dysfunction, large doses of potassium penicillin G given intravenously may result in electrocardiographic changes indicative of hyperkalemia, and if the situation is not corrected, cardiac arrest may eventuate. Each 15 million units of aqueous crystalline penicillin G supplies 975 mg. (25 mEq.) of ionic potassium. At present the calcium and aluminum salts of penicillin G are seldom used in the United States. Similar considerations are required on use of the semisynthetic penicillins. For example, in the management of patients having heart failure or renal dysfunction, one should recall that each 4 gm. of methicillin supplies 230 mg. (10 mEq.) of ionic sodium.

Penicillin G, especially when given in large amounts for prolonged periods, rarely has caused anemia, evidently reflecting immune erythrocyte destruction.[45] Granulopenia and anemia have occurred after administration of methicillin. Oxacillin has caused transient leukopenia. Some of the penicillins have been accused of other forms of hematotoxicity on occasion, but these contentions are more difficultly established.

Penicillin G, when given in large amounts, may cause cortical irritation, including convulsions, especially in patients having decreased renal function. Chronic brain damage may result. Since penicillin G in addition has precipitated acute psychosis and since anaphylactic reactions also have resulted in chronic brain damage,[46] perhaps one should be more aware of the potential neurotoxicity of the drug.

Whether methicillin has nephrotoxic effects is uncertain at this writing. Penicillin G rarely appears to have been nephrotoxic, evidently the toxicity being via the mechanism of allergic vasculitis.

During therapy with ampicillin, oxacillin, and cloxacillin, the serum glutamic-oxaloacetic transaminase activity may be increased, but the exact significance of this is not clear.

Phenethicillin, oxacillin, cloxacillin, and nafcillin are not well absorbed after oral administration unless the stomach is otherwise empty. Food interferes with the absorption of these drugs, and consequently the decreased levels in serum might be insufficient to eradicate an infection.

Use of the penicillins may depress the coccal members of the body flora and encourage the development of bacteremia, urinary infection, pneumonia, and other infections caused by overgrowth of resistant organisms such as gram-negative bacilli and Candida. Periodic examination of the sputum and stools may allow detection of changes in the body flora in time to avert clinical superinfection. As an example of this complication, in series of patients treated with methicillin, as many as a third have experienced superinfections due to gram-negative bacilli; fatal bacteremia due to gram-negative bacilli has occured.

As aforementioned, strains of Staph. aureus isolated from clinical material in British hospitals have demonstrated an increasing incidence of resistance to methicillin.[28,29] These strains may show a corresponding increase in resistance to other commercially available penicillinase-resistant semisynthetic penicillins.[27] The presumption is that when these drugs have been employed in a given environment for a period of time, strains of staphylococci will demonstrate increasing resistance to their action, as has occurred in Britain. This ominous possibility should be sufficient to militate against indiscriminate use of these drugs.

The serum concentrations of all the penicillins, both natural and semisynthetic, may be increased on conjoint administration of probenecid (Benemid®),[47] which blocks the renal tubular excretion of these antibiotics. However, a number of patients are unable to tolerate probenecid because of gastrointestinal irritation. Also, some practitioners do not favor the use of probenecid because its side effects may be confused with those of the penicillins or with manifestations of the infection being treated. Such side effects of probenecid include dermal reactions, anaphylaxis, fever, chills, and anemia. Of course, concomitant administration of probenecid in infections of the urinary tract would not be advisable, since decreased urinary concentration of the antibiotic would be likely.

CONCLUSIONS

Infections that previously responded to the naturally occurring penicillins G and V continue to respond, except those due to certain staphylococci. These penicillins continue to be the drugs of choice in many infections, including those due to pneumococci, streptococci, and meningococci, as well as in such conditions as syphilis, actinomycosis, and fusospirochetosis.

The semisynthetic phenethicillin and penicillin V are used more or less interchangeably. Of the penicillinase-resistant drugs, oxacillin, cloxacillin, and nafcillin are more active by weight than methicillin and can be given orally as well as parenterally. Serum levels of cloxacillin may be twice as high as those of oxacillin. Nafcillin is sometimes preferred because of its more persistent serum and tissue levels. Ampicillin has been effective in serious infections due to gram-negative bacilli, including influenzal meningitis and bacteremia due to E. coli.

Long-term evaluation is required in order to assess the comparative advantages of new antibiotics and the continued value of older ones. This is so, because the benefits as well as the untoward effects of a drug may not become known until several years have elapsed after its clinical availability. Their untoward effects, which may become apparent on further observation, may influence considerations regarding the ultimate clinical application of the individual semisynthetic penicillins.

The increasing number of effective penicillin derivatives and the diversity of their clinical

application, as well as their potential untoward effects, make it incumbent on the practitioner to be familiar with these drugs and to utilize them in a selective manner.

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Heart-Lung Bypass

Perspective and Present Status

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THE DEVELOPMENT of extracorporeal circulation and heart-lung bypass as it exists today was preceded by over one hundred years of laboratory effort. As far back as 1813, a concept of an artificial circulation was proposed (LeGallois).[1] In the early nineteenth century, physiologists were able to reinstate responses in organs by perfusing them with blood. In the late nineteenth century, machines for continuous perfusion were developed, but their clinical use awaited knowledge in understanding blood incompatability and coagulation. In 1937, Gibbon[2] introduced the concept of heart-lung bypass as an aid to cardiac surgery under direct vision. This paper was followed by a period of intense investigational work, guided by the idea that a machine of large capacity was needed for whole-body perfusion (Bjork).[3] The breakthrough was finally accomplished by advances in biologic technology in this century which permitted the construction of a practical machine. Such advances include the discovery of heparin and protamine, the development of physicochemically inert tubing and containers, and the introduction of antifoaming substances. The work of Andreasen and Watson[4] (1952) gave the necessary impetus to the application of heart-lung bypass in the human. They showed that even a limited flow of blood such as that furnished by the amount of blood draining from the azygous system during caval vein occlusion would permit survival after a drastic reduction of body perfusion lasting up to thirty minutes. This principle that a small perfusion flow directed mainly to the brain and the heart would suffice to prevent irreparable damage was successfully applied by Lillehei[5] (1955). Other highlights of this period were the correction of pulmonic stenosis using total bypass by Dodrill[6] (1952) and the first successful open intracardiac procedure under total cardiac bypass, the closure of a secundum atrial septal defect by Gibbon[7] in 1953. In the last ten years, the focus of interest in clinical perfusion has shifted many times. The inadequacy of arterial blood flow through a femoral vessel, the toxicity of high oxygen tensions, the danger of combining heart-lung bypass and hypothermia, the prevention of hemolysis, the maintenance of body blood volume, and so on, have each in turn been held responsible for clinical failures which were difficult to explain. This shifting of emphasis on various aspects of clinical perfusion indicates the necessity for maintaining a formal approach and for keeping to the fundamental knowledge of physiology which is known and which will be learned in the future in order to solve the problems of extracorporeal circulation.

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THE HEART-LUNG MACHINE (PUMP OXYGENATOR)

An ideal artificial heart-lung machine must be simple and dependable. The oxygenator should oxygenate 4 to 5 liters of venous blood per minute, adequately eliminate carbon dioxide, have a reasonable priming volume, and produce minimal blood damage. The blood pump should have a minute volume ranging from 200 to 5,000 ml. against pressures up to 180 mm. Hg, an adjustable stroke volume and pulse rate, and a linear relationship between pulse rate and output. The operation should be automatic as well as manual with minimal damage of blood elements and easy maintenance. It should be able to permit normal autoregulation throughout the organism.

A good heart-lung machine not only should have the ability of rapid introduction of oxygen into and elimination of carbon dioxide from 4 to 5 liters of collected blood but also should have the ability to separate the liquid phase from the gas phase and to infuse the blood under pressure back into the organism within one minute. The

process of oxygen uptake in an oxygenator differs from the corresponding process in the natural lungs. In the natural lungs, a monocorpuscular film of blood is exposed to a partial pressure of oxygen of 100 mm. Hg of 0.75 plus or minus 0.25 seconds, but in the artificial oxygenator, a relatively thick layer of blood comprising 20 to 60 layers of erythrocytes is exposed to a partial pressure of oxygen of about 700 mm. Hg for many seconds. Turbulence is the major factor in enhancing the admixture between the inner and outer layers of the blood film. The blood film is much thicker than in the pulmonary capillaries, thus interfering with the red cell exposure to oxygen. This defect can be partially compensated for by mechanical stirring and prolongation of contact time. The carbon dioxide (CO_2) elimination is adequate in most designs of heart-lung machines as long as there is no defect in oxygenation, but the "membrane," "small bubble," and "foam" type of gas exchange devices may favor CO_2 retention in the presence of perfect oxygenation. Today, there are three types of oxygenators now being used in laboratory and clinical practice: bubble oxygenators, film oxygenators, and membrane oxygenators. Among these, the bubble oxygenator designed by DeWall and the rotating disk oxygenator designed by Kay-Cross are most commonly used. The normal human adult lung has a surface area of about 100 square meters available for alveolar-capillary gas exchange. Fortunately, much less area is needed in oxygenators because the blood is exposed during bypass to 100% oxygen instead of 20% (the amount in air) and because the patient is in a subbasal state. Oxygen diffuses much less rapidly than CO_2, and for this reason the design of oxygenators attempts to get a large surface area in a small volume. This may be obtained by the types of oxygenators mentioned above. Each accomplishes this in its own way and each has its advantages and disadvantages.

Arterialization of venous blood by oxygen bubbles was proposed by Von Schroeder in 1882. Owing to the inability of removing the excess of gas bubbles from the blood stream, this principle of arterialization was never successfully used either in animals or human beings until 1950, when Clark and associates[8] introduced silicone compounds as defoaming agents. The oxygen is dispersed into the venous blood through small holes producing blood foam where the mixing of blood and oxygen occurs and CO_2 is eliminated. The oxygenated blood is then defoamed by exposing it to surfaces coated with suitable silicone. Perfusion can then be carried out without involving the risk of gas embolization. The flow of oxygen gas must be at least 25 times larger than the oxygen uptake. The usual ratio of oxygen flow to blood flow is two to three to one. The amount of gas flow, the size of the bubbles, and the composition of the gas mixture are three important factors in bubble oxygenators. Small bubbles produce good oxygenation, but they interfere with CO_2 elimination, are more difficult to remove, and may cause more hemolysis. Large bubbles are less efficient in oxygenation but they are good for CO_2 elimination and easy to be removed from the bloodstream. Considerable interest recently has been expressed in oxygen poisoning as a cause of some of the unexplained difficulties with perfusion (Effler,[9] 1959). The damaging effect of an atmosphere of pure oxygen on metabolism has been demonstrated, and there is evidence of damage to the lungs in mammals, after a number of hours' exposure to oxygen. A possible source of difficulty can be envisioned if it is noted that the temperature of the bypass apparatus and its contained blood approaches room temperature unless efforts are made to keep it higher. Oxygen is more soluble in cold than warm blood and if blood is pumped back to a warmer subject, oxygen will come out of blood and exert a higher partial pressure, even in the range known to cause oxygen poisoning. The role this plays in clinical perfusions is still unsettled. Gaseous oxygen has also been implicated as a possible source of bacterial contamination.

The bubble oxygenators consist of three main parts—a bubbler for oxygenation, a defoamer, and a blood settling chamber for separation of blood from the gas phase. The last two chambers are frequently combined. A sigmamotor pump was originally used. The details are described by DeWall and associates.[10] Bubbler oxygenators are extremely efficient oxygenators; their safety depends on the reliability of the debubbling process. Film oxygenators bring about a large blood-gas interface by spreading out a swiftly moving stream of blood into a relatively thin film. Gas exchange is then facilitated by exposing the blood film to an atmosphere of oxygen. The thickness of the film obtained by gentle spreading of blood over a solid surface is by necessity greater than the diameter of the pulmonary capillaries. Arterialization of blood remains rather inefficient in these kinds of oxygenators because the superficial layer of blood film acts as a barrier to gas exchange between the atmosphere and the subjacent layers. This defect usually can be compensated by marked gas tension differences between the blood film and the adjacent atmosphere, by prolonged time of expo-

sure of the film, and by turbulent flow to constantly renew the exchange surface. In membrane oxygenators, the blood film and gaseous oxygen are separated by a semipermeable membrane. Gas transfer across the membrane depends upon the nature, thickness, surface, and degree of hydration of the membrane as well as the partial pressure differences of the diffusing gases on opposite sides of the membrane. The main disadvantage of the membrane oxygenator may be inadequate CO_2 elimination.

The heart of the average human adult can pump up to 30 liters per minute of blood during exercise. Since blood pumping systems are used exclusively for the perfusion of organisms under resting conditions, the "desired" volume flow corresponds to the minimal performance of the human heart (about 4.5 liters per minute). The operation of the pump with high flow velocity is very detrimental to blood. The oxygenator, the pump, and, especially, the suction are most traumatic to blood. The arterial return introduces problems of pressure and flow. During heart-lung bypass, we have a relatively small cannula inserted into one of the arterial branches (subclavian or femoral) resulting in high resistance to flow and more trauma to blood due to turbulence at the cannula outlet. Denaturation of plasma proteins, hyperlipemia leading to sludging in the capillary beds, hemolysis of red cells, and large platelet and fibrin aggregates which may act as emboli in the pulmonary vascular bed have been described. The DeBakey twin roller pump and the multiple finger pump are commonly used today; the latter is noisy and rather traumatic to blood. In the past, the most controversial point concerning pumping is whether pulsatile flow is more desirable than nonpulsatile flow. In classic physiology, the pulse pressure is felt to be important in maintaining normal organ functions, especially for the kidney. Up to now, no conclusive evidence has been uncovered to show that at the organ level pulsatile flow is superior to constant flow at the same mean pressure.

HEMODYNAMICS AND METABOLIC ASPECTS OF EXTRACORPOREAL CIRCULATION

During total body perfusion, the organism is anesthetized, the heart and lungs are empty, and the superior and inferior caval beds are usually separated. The distribution of blood among the organ systems is altered because of exclusion of the heart and lungs and the manner of drainage and the infusion of the blood. The extracorporeal flow is often below the resting cardiac output. During perfusion, the hemodynamic goal is to make the bypass procedure deviate minimally from normal physiologic function, that is, to obtain a steady flow rate approximating the cardiac output of the patient before perfusion. This aim is approached by the maintenance of a normal blood volume and adequate flow through sufficiently large venous cannulas and a properly working pump. Under these conditions in the patient, the commencement of extracorporeal circulation initially results in the main aortic pressure dropping 10 to 30 mm. Hg below the value before bypass. The main central venous pressure is ordinarily maintained between 5 and 15 mm. Hg, a level somewhat higher than before surgery. The initial slight drop of arterial pressure at the beginning of bypass may be due to the fact that systemic flow before bypass is lower than that mechanically maintained when bypass is started, at which time additional blood is required to fill the system for the higher flow. In trouble-free perfusions, the arterial pressure tends to return to normal and, after bypass, increases to a moderate extent (Dodrill[11] 1957, Anderson and Senning[12] 1958, and McGoon[13] 1960). The arterial pressure is approximately a linear function of perfusion rate (Kirklin,[14] 1957, and Grodins[15]). The peripheral resistance in successful perfusions tends to increase gradually compensating for some degree of vasodilatation at the beginning of bypass (McGoon,[13] 1960). Thus, only minor vasomotor changes occur during uneventful perfusions. When the perfusion is prolonged, a second pronounced vasodilatation occurs (Sanger,[16] 1960), caused by a host of hemodynamic and metabolic derangements. The mechanically supported circulation also reacts differently than the intact circulatory system. The former is extremely sensitive to small variations in the blood content of the vascular system. The latter can readily compensate for moderate amounts of transfusion or blood loss. The maintenance of a constant systemic blood volume then becomes an essential condition for smooth perfusions. Thus, the monitoring of blood volume assumes major importance. With no practical way of detecting body blood volume directly and immediately at any one point during bypass, indirect techniques must be employed. Most methods of controlling the body blood volume rely upon frequent measuring of the volume of blood in the extracorporeal circuit. The sum of the body blood volume and the extracorporeal blood volume is constant so that the change in one direction in one reflects a change in the opposite direction in the other. Donald and Moffitt[17] (1957) stated that in dogs a 10% reduction in blood volume causes a 50% fall in venous re-

turn to the extracorporeal circuit and consequently a 50% fall in arterial infusion flow. Herron[18] (1958) demonstrated a direct relationship between body blood volume and available venous return. At 100% of control blood volume, the flow rate in dogs averaged 39 ml. per kilogram per minute; to obtain flow near normal cardiac output, it was necessary to expand the blood volume to 130% of the control. In man, this tendency to blood pooling is usually absent and heart-lung bypass can be carried out without increase in systemic blood volume. McGoon[13] (1960) had demonstrated a decrease in blood volume at the start of perfusion of about 20%, due to emptying of the heart and lungs. Replacement of this blood is necessary should the venous flow diminish from blood loss or other cause. Kirby[19,20] (1959-1960) has shown that there is a constant intravascular blood volume for each perfusion rate and that there is a need for a larger circulating blood volume with increasing flow. Since the venous side of the circulation contains 80% of the body blood content, it is understood why venous pressure is closely related to systemic blood volume during (but not necessarily after) perfusion. The maintenance of a constant systemic blood volume is a major key to successful perfusions.

Under normal conditions, the various organ systems do not receive an equal share of the cardiac output. Some are profusely irrigated and others much less than one would expect from their weight or metabolic activity. The kidneys, for instance, receive 25% of the cardiac output but use only 8% of the total oxygen uptake, a renal A-V oxygen difference of only one to three volumes' percent. The heart, about the same weight as the kidneys, receives only 5 to 8% of the cardiac output, but since it needs more oxygen, its coefficient of oxygen utilization is high. Consequently, coronary venous blood is markedly desaturated. The distribution of blood in the intact animal and its redistribution after the institution of heart-lung bypass are dependent upon autoregulatory mechanisms of blood flow which are different for the various vascular beds and are not completely understood. However, the first determinate of regional flow is the vascular resistance best understood from a pressure flow relationship (Swan,[21] 1951). This relationship presented as a graph demonstrates a different curve and slope of curve for each organ, indicating that variations are not equal on a purely hemodynamic basis. Blood gas tensions in the perfusing blood play an important part, but here again, each organ has different mechanisms for compensating for such changes. These observations have evolved into a concept of selective and protective distribution of blood flow so that more vital tissues are selectively favored over less vital tissues, especially when flow rates are low and the amount of blood circulating is reduced. A fair amount of experimental work supports this view (Halley and Creech[22,23] 1958-1959, Reemstma and Creech[24] 1959, Brown and Smith[25] 1958, Shumacker[26] 1960, and Andersen[27] 1961). These studies essentially compared flow to various vascular beds under basal conditions to those obtained in normothermic perfusions. The flow rates were arbitrarily classified as high (2.4 liters per square meter per minute), medium (1.8 liters per square meter per minute), or "azygous" (0.5 liters per square meter per minute). The brain is best protected and the heart next. Renal flow drops markedly with decreasing flow rates with a delayed drop in renal oxygen uptake. The splanchnic bed is fairly well protected, and deprivation of flow does not occur unless the flow rate is very low.

The question of "high" or "low" flow has been present from the first. The low flow concept was given early impetus by the demonstration of the azygous principle in the laboratory as mentioned above and the development of controlled cross-circulation. The ideal of most of the pioneers such as Gibbon, however, was a bypass flow equal to that of the patient before bypass. The present trend is to return to this ideal now more safely achieved with better equipment. The resting flow of 2.5 liters per minute, which is a normal cardiac index under the conditions of a normal chest and an anesthetized patient, can be expected if there is no obstruction to venous outflow. The usual perfusion rate, in terms of liters per square meter per minute, varies with the size of the patient: 2.4 liters per square meter per minute when the surface area is about 1.5 square meters, 2 liters per square meter per minute when the surface area is 1 square meter, and 1.8 liters per square meter per minute in the average adult. Kirklin (1958) and associates[28] recommended flow rates 2.2 to 2.5 liters per square meter per minute regardless of age.

The basic work of Bigelow[29] (1950) showed that the oxygen consumption of warm-blooded animals can be lowered without incurring an oxygen death. This prompted the idea that cooling might be usefully combined with heart-lung bypass especially when perfusion rates were low. It was feared that such combined procedures would compound difficulties inherent in each.[30] The work of Gollan,[31] Dripps,[32] DeLorme,[33] and others, however, added credence to the idea of clinical applicability of extracorporeal cooling.

Hypothermia can be accomplished by cooling the blood in an external circuit (core cooling) or by immersion of the body in ice water (peripheral cooling). Both have application today, but core cooling accomplished by efficient heat exchanges are most commonly used with heart-lung bypass. Bloodstream cooling gives a larger metabolic saving than does peripheral cooling because the blood preferentially reduces the temperature in the organs which have the highest blood flow and the largest oxygen consumption—kidney, heart, brain, and liver. Also, rewarming is more quickly achieved returning the vital organs to normal function earlier than the less important ones. Anesthesia can be kept at a minimum because of the absence of reflex shivering during the cooling phase. Oxygen requirements are diminished with hypothermia, but since they cannot be predicted, the venous oxygen saturation should be kept within a normal range.

The major disturbance of acid-base balance resulting from extracorporeal circulation is metabolic acidosis. There is a moderate depression of plasma bicarbonate accompanied by an elevation of blood lactic acid. The acidosis, associated with heart-lung bypass procedures, is of hypoxic origin and develops because the perfusion fails to distribute as much oxygen as the tissues require. Low or decreasing blood flows are associated with progressive decreases in blood pH; while at higher flow, the pH remains relatively stable. If the patient has inadequate tissue perfusion during cardiopulmonary bypass and is unable to maintain a high enough cardiac output after bypass to clear the accumulation of acid metabolites, he will develop hypotension together with acidosis and finally succumb. Other adverse perfusion factors are those which result in a decreased oxygen carrying capacity such as insufficient saturation of the arterial blood by the oxygenator or excess of dilution of the blood with saline or glucose. Other causes of metabolic acidosis are sustained arterial hypotension, a depressed state from anesthesia, and surgical trauma and "hyperventilation" (10 liters per minute of oxygen [O_2] is sufficient) of the blood in the artificial lung resulting in a shift of the oxygen dissociation curve toward a region less favorable to oxygen release from the oxyhemoglobin to the tissues.

Bypass itself alters fluid and electrolyte balance. On the anionic side, plasma levels of lactate pyruvate, inorganic phosphate and chloride increase and bicarbonate decreases. On the cationic side, sodium increases and potassium and calcium decrease. The effects of the potassium and calcium changes on the myocardium tend to cancel out. Water is lost from exposed surfaces, but during long perfusions there is a tendency to retain water so that edema may result.[30]

The problem of which parameter should be used to monitor the adequacy of perfusions remains unsettled. One may rely upon hemodynamic guides such as flow-rate equal to or near basal cardiac output, maintaining an arterial pressure above 70 mm. Hg, or a central venous pressure between 5 and 15 mm. Hg. Metabolic measurements may be employed such as arterial oxygen saturation between 95 and 100%, venous oxygen saturation of 70%, an arterial blood pH between 7.25 and 7.45, and an arterial carbon dioxide tension between 32 and 42 mm. Hg. In addition or in combination with any of the above, organic signs may be followed such as the electroencephalogram and the excretory function of the kidney. One practical solution to the control problem is to monitor only those parameters which empirically have been found useful in the operating room, such as arterial, superior, and inferior caval pressures, electroencephalogram, blood pH, and so on.

THE PERFUSATE

Many new clinical problems have appeared as a result of the introduction of extracorporeal circulation in cardiac surgery. The heart-lung machine was originally primed either with fresh heparinized blood or anticoagulant acid citrate dextrose (ACD) blood with the addition of heparin and calcium ion (Foote and associates[34] and Zuhdi and associates[35]). The difficulties in using these primes—procurement of large amounts of blood, the possible transfusion reaction, the transmission of diseases, and the occurrence of the homologous blood syndrome (Gadboys and associates[36])—are drawbacks in the ultilization of whole blood.

The clinical use of hemodilution for total body perfusion without the addition of priming blood was introduced in 1959 by Panico and associates[37] and recently reported by Zuhdi,[38] Cooley,[39] and DeWall.[40] The obvious virtues of hemodilution include reduction in blood requirement, a decrease in the hazard of transfusion reaction, and reduction in blood viscosity allowing for better perfusion at lower temperatures. The possible disadvantages are lower concentration of the blood elements, which may reduce the oxygen carrying capacity of the blood, and alterations in fluid and electrolyte patterns. Five-percent dextrose solution is quite acid, having a pH of 6.7, and it is mildly hypotonic with regard

to erythrocytes. Five percent dextrose in 0.2% saline will minimize hemolysis (Linder and associates[41]). Long and associates[42] used a low-molecular weight dextran in extracorporeal circulation to improve the perfusion. He reports a significant decrease in cellular aggregation and destruction of red cells and platelets. Hellström and Björk[43] used low-molecular weight dextran in 10% dextrose, or dextran 40 (Rheomacrodex®), in the priming of their heart-lung machine in place of one unit of blood. They report a significant reduction of thrombocytes and a slight but statistically significant increase in postoperative bleeding. Hepps and associates[44] used a hemodilution technique with a balanced electrolyte solution or 5.6% low-molecular weight dextran in an electrolyte solution in dogs; they reported improvement in results and amelioration in the pulmonary postperfusion syndrome. Roe and associates[45] reported satisfactory results in patients with hemodilution during extracorporeal circulation using a balanced isoosmolar electrolyte solution. The degree of hemodilution is usually limited to 50% or to a hematocrit of 20. It has been necessary to add small amounts of blood in some patients, particularly children. The hemodilution principle is gaining wider acceptance at the present time.

POSTOPERATIVE CARE

The physiological derangements can be far more extensive in cardiac cases with cardiopulmonary bypass than other surgical procedures. Clowes[46] (1960) and Moore[47] (1963) emphasized the disturbances of acid-base balance during and after bypass. Lee and Maloney[48] (1961) suggested that the denaturation of plasma protein is a cause of morbidity and death after intracardiac operations. Pulmonary injuries following extracorporeal circulation have been reported both in dogs and man by numerous workers: Dodrill[49] (alveolar collapse), Schramel[50] (hemorrhagic atelectasis), Neville[51] (pulmonary vasculitis), Ellison[52] (pulmonary membrane injury), and Rossi[53] (platelet and fibrin emboli). All of these factors in addition to the cardiac disease will increase the work of breathing and aggravate oxygen deficit. They contribute to the difficulties in postoperative management. Clinical observation alone is not sufficient for such patients. Careful monitoring of blood pressures, blood gases, pH, cardiac output, and electrocardiogram and the use of an efficient volume controlled respirator are all very important for the safety of the patient.

One of the most common complications following open-heart surgery is acidosis, either respiratory or metabolic in origin. The former is due to respiratory insufficiency and the latter is due to inadequate perfusion. Respiratory insufficiency occurs either when the patient cannot perform sufficient respiratory work to provide adequate ventilation or when the functional capacity of the lung has been reduced significantly. Both factors usually play a role and the result is a lowered arterial oxygen tension and an accumulation of carbon dioxide resulting in respiratory acidosis. Acidosis from any cause has an adverse effect on the myocardium, that is, a decrease in cardiac output, a depression of response to stimulative drugs, and the appearance of arrhythmias.

To protect the patient from acidosis, the arterial carbon dioxide pressure (pCO_2) and the arterial pH levels should be followed carefully. If the pCO_2 remains within normal limits and the pH falls (metabolic acidosis), sodium bicarbonate or tris (hydroxymethyl) aminomethane (THAM) is given to the patient in sufficient doses to bring the pH back to normal. THAM has the drawback that it has been associated with respiratory embarrassment. If the arterial pCO_2 rises (respiratory acidosis), the fault is pulmonary insufficiency, and measures to improve ventilation must be taken, such as high oxygen breathing, removal of secretions, tracheostomy, and the use of a respirator (Spencer[54]).

The most common cardiac manifestations arising from a low arterial oxygen tension are arrhythmias and increased ventricular irritability. Cardiac arrhythmias usually result in a decrease in cardiac output, decreased coronary perfusion, and increased myocardial oxygen debt and may lead to death in severely ill patients. Assisted ventilation by use of a mechanical respirator will decrease the work of breathing and increase the blood oxygen tension, thus reducing cardiac work. It is an important measure to prevent or control the respiratory insufficiency in the immediate postoperative period.

In the operating room, blood volume is restored to the preoperative level as closely as possible by the administration of fresh citrated blood. This is aided by a close observation of blood balance during the procedure and by weighing the patient before and after operation. The measurements of blood volume in the intensive care unit are helpful in demonstrating losses not otherwise evident. Bahnson and Spencer[55] leave small polyvinyl catheters in the pulmonary artery and left atrium. Cardiac output can be estimated by oxygen saturation of the mixed venous blood and adequacy of pulmonary

ventilation determined from the left atrial sample. The left atrium pressure and blood volume can be measured and together with weight changes, central venous pressures, mixed venous oxygen content, and pH (in the absence of hypoventilation low pH indicates a low cardiac output) are helpful in determining limits to which transfusions might be pushed in the presence of a failing myocardium. As regards blood volume, Yu and associates[56] have shown that a tagged, red-cell technique may be preferable. The patient is left in an atmosphere of high oxygen and humidity for several days. Tracheostomy is at times lifesaving and is frequently employed when secretions are profuse and respirations embarrassed. In many instances a respirator is temporarily employed; in some instances it has been successfully used for weeks.

Heart blocks occur less frequently than in the earlier years of open-heart surgery. In many instances, transient blocks occur with no long-lasting effects. Atropine, isoproterenol (Isuprel®), and ephedrine are drugs helpful in this situation, but when any question exists about the cardiac rhythm at the end of the operation, an electrode wire is left sutured to the myocardium.

All patients have preoperative blood clotting studies. These studies are usually normal. Despite this and meticulous hemostasis, postoperative bleeding can be a problem, especially in some of the older patients with tetralogy of Fallot where the hematocrit is in the 70 to 80% range. Reexploration is necessary when bleeding is sudden and massive or particularly if there appears to be cardiac tamponade.[55] For the last reason, it is performed most frequently. If bleeding persists and clotting is inadequate, epsilon aminocaproic acid has been used in several cases with some benefit.

If a patient whose blood pressure is stable, hydration adequate, and urinary outflow tract patent develops oliguria (urine flow less than 20 ml. per hour), mannitol therapy with a test dose of 12.5 gm. administered intravenously in a 25% aqueous solution is indicated. If no response is obtained after the initial therapy, acute renal shutdown is suspected. If urine flow increases within the next three hours but does not exceed 40 ml. per hour, a second dose is given, (Barry and Malloy,[57] 1962). A urinary sodium concentration of 50 mEq. per liter is indicative of acute renal shutdown. If urine flow exceeds 40 ml. per hour in response to the test dose, mannitol infusion is titrated against the urinary output to maintain the urinary flow at about 100 ml. per hour. On many chest surgical services, mannitol is used prophylactically during heart-lung bypass, but despite a marked diuretic effect, a protective action on the kidneys has not been proved.

The fluid intake should be restricted to 500 ml. per square meter of body surface area per twenty-four hours, plus additional fluid to equal the urinary output and the gastrointestinal and blood loss. Sodium intake should be limited to 50 mEq. per square meter per day of body surface and potassium to 40 mEq. per square meter per day. All depends upon the condition of the patient. After the first two days, fluids will usually be tolerated by mouth, making fluid and electrolyte regulation easier. Blood is replaced volume per volume.

Body temperature should be kept below 100° F. (96.8 to 100° F.) either with external cooling or administration of acetylsalicylic acid, or both, to maintain lower metabolic rate in order to decrease the cardiac burden. Severe shivering due to external cooling should be avoided but slight shivering during rewarming contributes to the return of normal temperature conditions. Urinary output and the specific gravity of the urine should be closely observed. Digitalis in adequate dosage is used as indicated to slow down the ventricular rate if the heart beat is too fast. Digitalis is not routinely given before operations, because of a higher incidence of arrhythmias during operations in fully digitalized patients and also because of uncertainty about loss of an indeterminate amount of drug in the blood of the pump system during bypass.

SUMMARY

The heart is the last organ of the body to be made amenable to surgical intervention for the correction of its defects and diseases. The introduction of extracorporeal circulation has made it possible for the surgeon to correct the intracardiac lesions in a bloodless field under direct vision. Successful open-heart operations are not possible without an understanding of the basic physiology of extracorporeal circulation and the metabolic alterations produced by disease and stress. The heart-lung machine is now quite safe for clinical use, but it is far inferior to natural organs. A safe heart-lung machine approaching the efficiency of natural organs is the product that will be obtained as our knowledge is expanded and as our understanding increases.

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Diagnosis of Primary Lung Cancer in the Aged Patient

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THE DIAGNOSIS of lung cancer is considered whenever a patient over the age of 45 complains of a change in his cough pattern or whenever his routine chest film shows a suggestive pulmonary shadow. The diagnosis is established when neoplastic cells are seen either in sputum or in surgically obtained specimens. To evaluate the guidelines in patients past 65, primary lung cancers found on autopsy during seven years in a custodial home and hospital for the aged were reviewed.

During the years 1959 to 1965 inclusive, 529 male patients died in the institution; 133 were autopsied and 5 primary lung cancers were reported. During this same time, 914 female patients died, 207 were autopsied, and again 5 primary lung cancers were reported. It will be seen from the case summaries (see Table) that the pulmonary symptoms were attributed to the neoplasm in 2 of the 10 patients. In 4, the complaints of cough, sputum, hemoptysis and dyspnea were ascribed to nonmalignant, preexisting disease, and in the remaining 4, no pulmonary complaints were offered at all. The X-ray interpretations are also summarized in the table. Of 5 patients admitted with abnormal chest films, only 1 was referred with the diagnosis of lung cancer, although the other 4 had lesions consistent with the presumptive diagnosis of primary neoplasm. Of 5 patients admitted without significant X-ray abnormalities, 2 developed X-ray lesions suggesting neoplasm on routine follow-up and 3 did not. The effectiveness of the search for neoplastic cells is seen in the same table. Tissue diagnosis of cancer was made in 3 of the 5 patients who accepted chest tap, bronchoscopy, or node biopsy. None had positive sputum cytology when spontaneously produced sputum was examined. Seven of the 10 patients had a correct premortem diagnosis made one to fifteen months before death. The diagnostic guidelines are only moderately successful.

In the aged, spontaneous complaints of a change in cough pattern are of minor value. Some patients deny all complaints because of their severe arteriosclerotic brain disease or because of severe deprivation depression (Figs. 1A and B). Other patients minimize their discomfort because they have suffered so long from emphysema, bronchitis, or persistent heart failure that cough, sputum, and dyspnea have no particular significance for them (Figs. 2A and B). Some patients offer complaints which are misleading because they arise from the metastatic focus rather than from the primary lesion (Figs. 3A and B). To improve the diagnostic value of symptoms in the aged, it is necessary that the character of the cough and of the sputum be observed and recorded by the hospital personnel much as the temperature is.

In the aged, the yearly survey chest X-ray has minor diagnostic value. The yearly interval was determined originally as a practical compromise between financial cost and the yield that could be obtained in a healthy population over the age of 45.[1] After the age of 65, not only is the cancer yield greater but the population is no longer healthy. Two of the 10 patients herein summarized had had a previous malignancy. The risk of multiple primary malignancies has been well documented.[2] Four of the 10 patients had old lung scars from previous tuberculosis, bronchiectasis, emphysema, or aspiration pneumonia. The risk of lung cancer in individuals with such scars is ten to twenty times that of the general population.[3] To improve the diagnostic value of the survey chest film in the aged, the yearly schedule must be shortened and made

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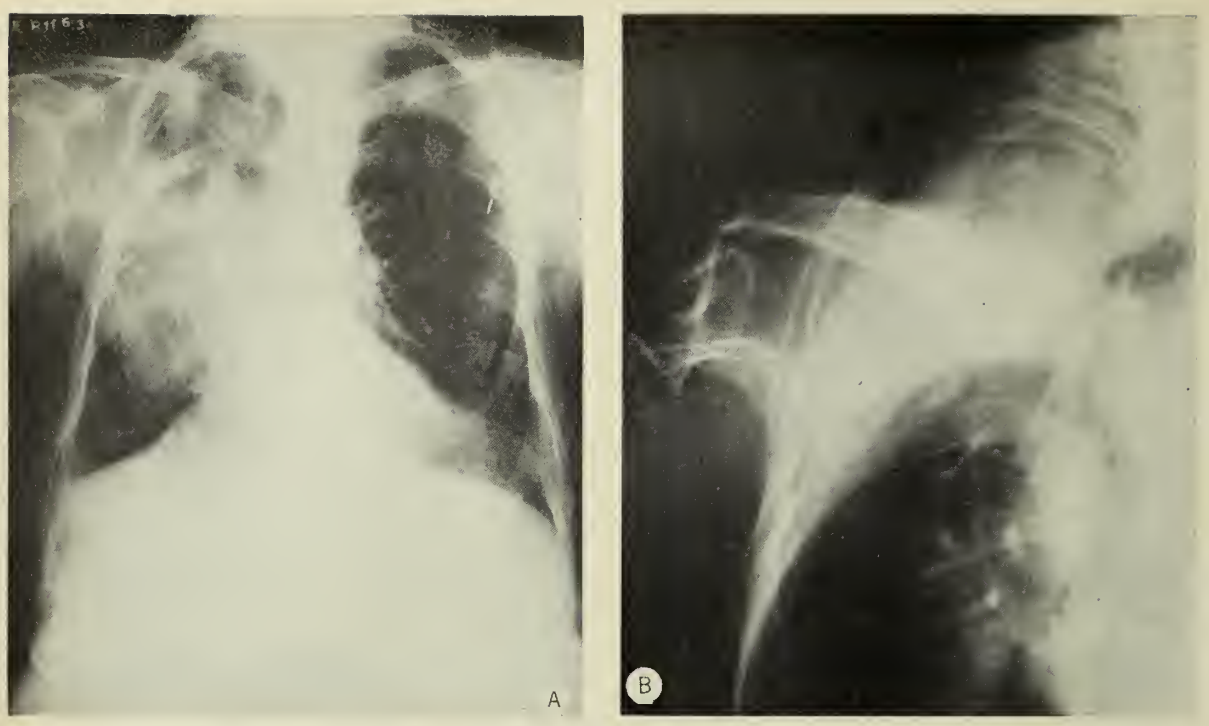


Fig. 1. [A] This mentally alert patient was admitted without complaints. Her chest film showed severe, right lung disease suggesting a pulmonary carcinoma. [B] Four months later she was still without complaints. Severe destruction of her right third posterior rib had occurred. Brain examination was not permitted; it is impossible to state whether her lack of symptoms was due to organic brain disease or to deprivation depression.

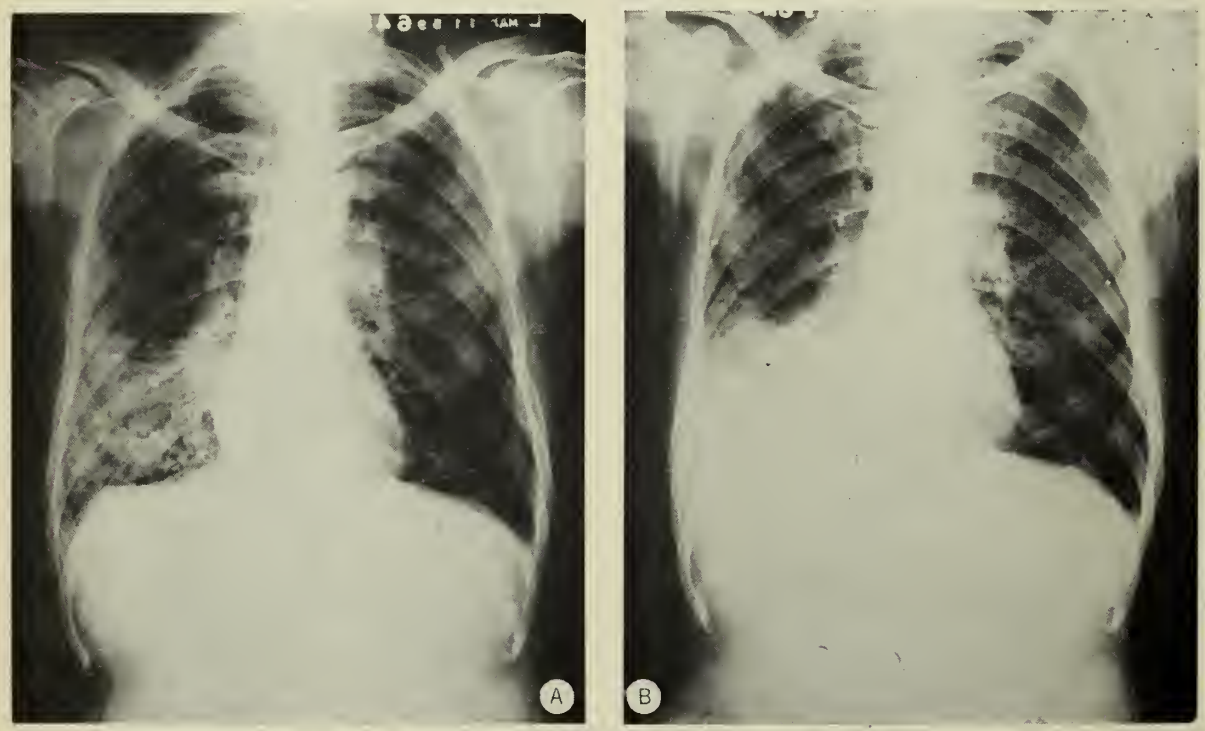


Fig. 2. [A] This patient had chronic cough, purulent sputum, and blood spitting for fifty years. His X-ray showed a few scattered infiltrations in both lungs, an exaggerated right lower hilus, and a condensation of infiltration at the right base. Pulmonary neoplasm and bronchiectasis were considered. [B] Six months later he had noted no change in symptoms except for increasing hemoptyses. His X-ray showed progression of the right basal lesion.

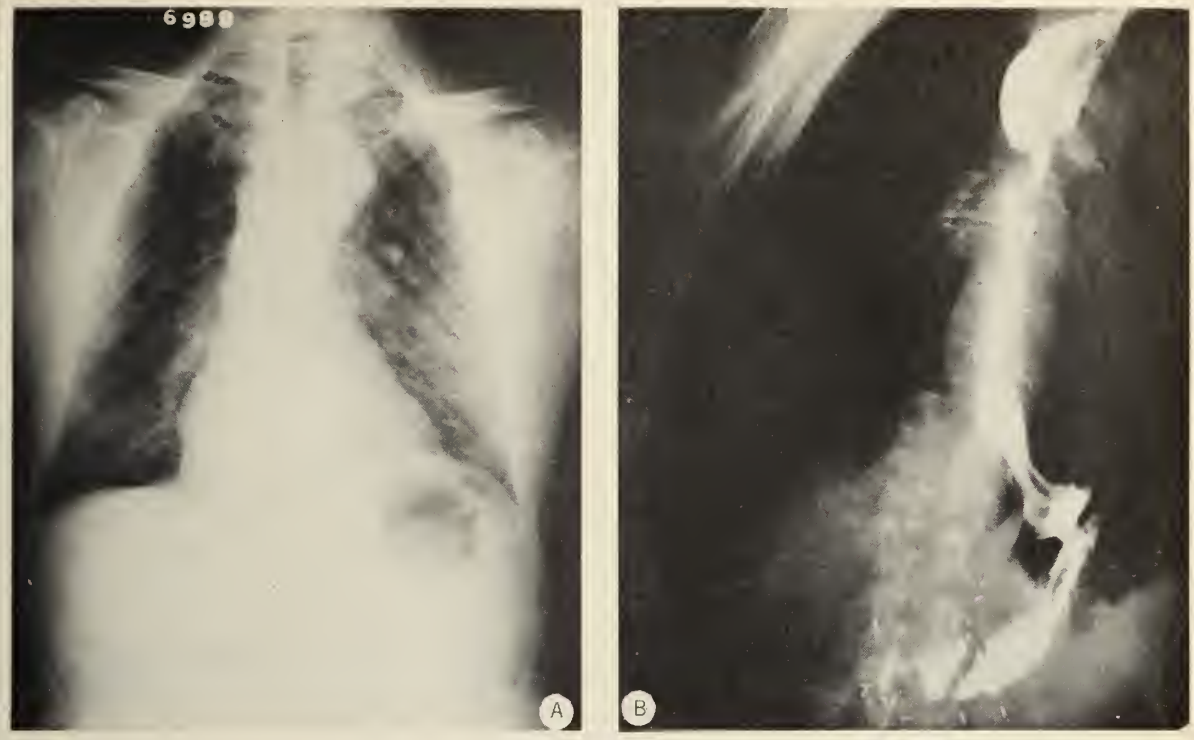


Fig. 3. [A] This patient was admitted without pulmonary complaints; his X-ray showed a Ghon tubercle in the left second interspace and bilateral apical thickening. His lesions were considered insignificant. [B] Severe difficulty in swallowing had developed; no pulmonary complaints were noted. The degree of esophageal abnormality was seen best in this X-ray. Clinical diagnosis: esophageal carcinoma. His lung fields remained unchanged.

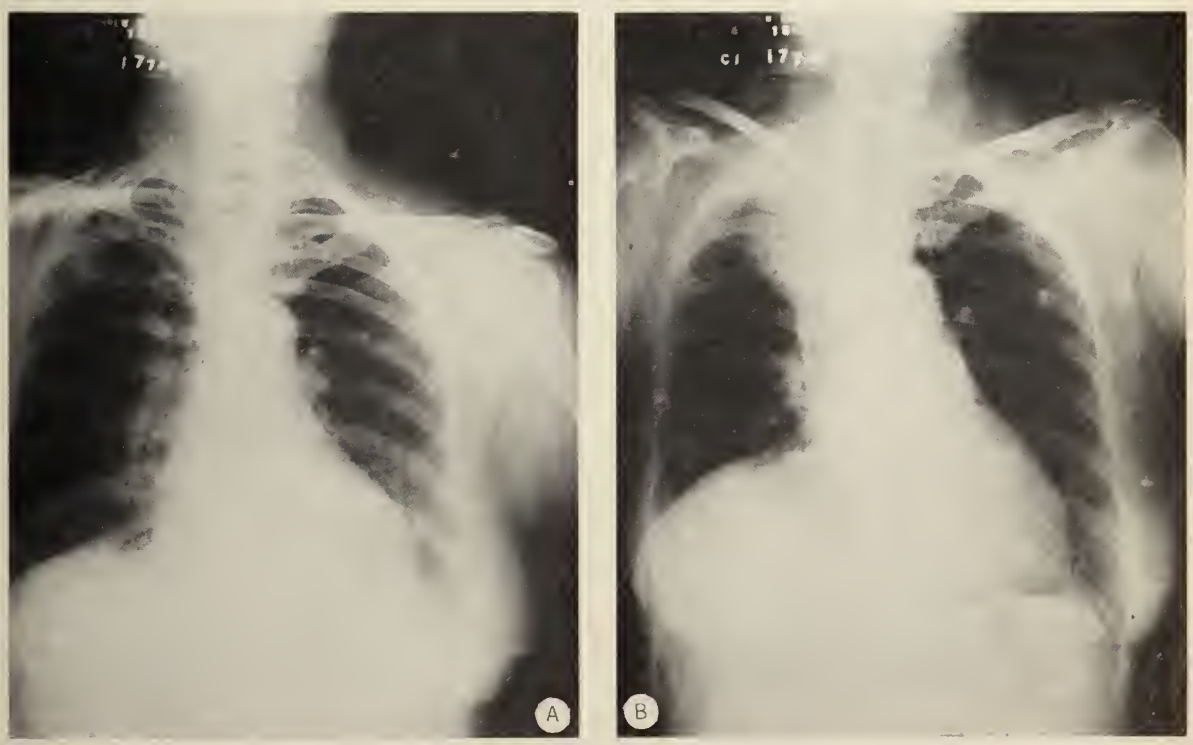


Fig. 4. [A] The patient had no pulmonary complaints; her admitting chest film showed slight exaggeration of the right upper hilus with soft infiltrates in the periphery of the right first and second interspaces. The diagnosis of pulmonary neoplasm was considered. [B] Two months later she still did not present pulmonary complaints, but a marked increase in her right upper lobe lesion with elevation of the right diaphragm was seen.

Case	Preadmission diagnosis	Admission Complaints	Admission X-ray	Course pertinent to pulmonary disease	X-rays during hospital stay	Clinical diagnosis of primary lung cancer: Suspected	Clinical diagnosis of primary lung cancer: Tissue diagnoses	Terminal diagnosis before autopsy	Autopsy diagnosis
Admitted 6/14/54, age 71 Died 9/19/63, age 80 Male	Arteriosclerotic cardiovascular disease, diabetes, severe cerebral arteriosclerosis	Weakness, confusion, petit mal	Negative	1959, first attack bronchopneumonia; 1960, increasing confusion requiring large amount sedation; 12/61, recurrent wheezing, dyspnea, and cough; pulmonary complaints twenty-one months before death, ascribed to aspiration	Transient bronchopneumonia 1959 on; impression, aspiration pneumonia	No	No	Arteriosclerotic cardiovascular disease, cerebral arteriosclerosis, aspiration pneumonia	Alveolar-cell carcinoma, organizing pneumonia
Admitted 4/18/56, age 81 Died 7/27/61, age 86 Female	Arteriosclerotic cardiovascular disease	Agitation	Negative	1960, cholecystectomy and thereafter increasing agitation requiring large doses sedation; pulmonary complaints one day before death, ascribed to pneumonia	Negative until 3/61 when cancer was suspected	Four months before death	No; 5/29/61, scalene node negative	Pulmonary carcinoma, arteriosclerotic cardiovascular disease, cerebral arteriosclerosis	Nondifferentiated bronchogenic carcinoma, organizing pneumonia
Admitted 5/9/59, age 74 Died 12/8/59, age 74 Male (See Figs. 2A-B)	Bronchiectasis past fifty years	Cough, purulent sputum, streaking	Abnormal	Recurrent fever, purulent sputum, increasing hemoptyses, and terminal pulmonary edema; pulmonary complaints fifty years before death, ascribed to bronchiectasis	Scattered lesions on admission, especially right base, cancer suspected; right base progressed; 5/59, cancer suspected	Seven months before death	No	Pulmonary carcinoma, bronchiectasis	Bronchogenic carcinoma, healed apical tuberculosis
Admitted 7/4/60, age 79 Died 11/13/63, age 82 Female	Arteriosclerotic cardiovascular disease, mild diabetes	Weakness	Negative	4/63, cough and fever one week with recovery; 11/9/63, dyspnea, cyanosis, rise in blood sugar and urea, going on to uremic coma; pulmonary complaints one month before death, ascribed to pneumonia	Negative until 10/9/63; impression, bronchopneumonia	No	No	Bronchopneumonia, arteriosclerotic cardiovascular disease, uremia	Atypical cells in fibrotic area, pneumonia, Mucor mycosis
Admitted 11/21/60, age 78 Died 3/18/61, age 78 Female (See Figs. 1A-B)	Arteriosclerotic cardiovascular disease, osteoarthritis, kyphoscoliosis	None	Abnormal	Progressive leg edema; 2/20/61, cough, weakness going on to increasing dyspnea; terminal pulmonary edema; pulmonary complaints one month before death, ascribed to cancer	Admission film suggestive of cancer	Four months before death	No	Pulmonary carcinoma, arteriosclerotic cardiovascular disease	Mixed squamous and adenocarcinoma
Admitted 3/30/61, age 75 Died 6/23/61, age 75 Male	1949, leg amputated for sarcoma; 3/60, adenocarcinoma of colon resected; arteriosclerotic cardiovascular disease	Weakness	Abnormal	Increasing weakness; 4/61, enlarged axillary node; 6/61, swelling over dorsal spine; terminal myocardial infarct; no pulmonary complaints	Admission film suggestive of cancer	Three months before death	Yes; 4/61 node and 6/61 spine swelling, both positive for cancer	Pulmonary carcinoma, arteriosclerotic cardiovascular disease	Squamous carcinoma, thromboembolism of lungs
Admitted 5/30/61, age 69 Died 6/4/62, age 70 Male (See Figs. 3A-B)	Arteriosclerotic cardiovascular disease	[illegible]	Negative	11/61, difficulty in swallowing, progressing to regurgitation of food; treated with radio therapy to esophagus 12/61 and 3/62; 4/62, pain right shoulder; no pulmonary complaints	Calcified tubercle left second space; 11/61, esophageal lesion; lung cancer not suspected	No	No	[illegible] carcinoma of right main bronchus infiltrating the esophagus, organizing pneumonia	
Admitted 6/12/62, age 83 Died 9/6/62, age 83 Male	Bronchogenic carcinoma diagnosed 6/61; Rx: radiation	Cough, hemoptysis	Abnormal	Attacks of prostatism and of recurrent pneumonitis; 9/4/62, incarcerated hernia, shock, and death; pulmonary complaints two years before death, ascribed to cancer	Admission film suggestive of cancer	Fifteen months before death	Yes; bronchoscopy positive 6/61	Pulmonary carcinoma	Bronchogenic carcinoma
Admitted 11/1/62, age 74 Died 2/7/64, age 75 Female	Arteriosclerotic cardiovascular disease, severe cerebral arteriosclerosis	Confusion, weakness	Negative	Progressive mental deterioration; 1/14/64, fell and hurt shoulder; thereafter stupor until death, no pulmonary complaints	Negative until 1/16/64 when left pleural effusion seen suggestive of cancer	Three weeks before death	No; pleural fluid negative for cancer cells	Pulmonary carcinoma, cerebral arteriosclerosis, arteriosclerotic cardiovascular disease	Alveolar-cell carcinoma with metastases to pleura
Admitted 6/22/65, age 66 Died 11/5/65, age 66 Female (see Figs. 4A-B)	4/64, colon resected for adenocarcinoma, arteriosclerotic cardiovascular disease, epilepsy	Weakness	Abnormal	8/18/65 (?), myocardial infarct; found dead in bed 11/5/65; no pulmonary complaints	Admission film suggestive of cancer	Five months before death	Yes; 9/16/65, negative bronchoscopy; 9/21/65, positive pleural fluid	Pulmonary carcinoma, arteriosclerotic cardiovascular disease	Adenocarcinoma

more flexible. Any patient observed to have a chronic cough or a stable pulmonary scar, or both, needs a chest X-ray every six months. As soon as either the cough pattern or the chest film changes, the patient must be X-rayed again within one to three months, depending on the individual circumstances. The interval of three months should not be lengthened, since the doubling rate of most primary cancers has been shown to be about ninety days.[4] This rate is determined by the cancer itself and not by the age or sex of the patient (Figs. 4A and B).

Once the possibility of lung cancer is considered, cytologic proof of diagnosis is necessary. Most of the elderly refuse bronchoscopy and other surgical diagnostic measures, but medical tissue diagnosis can now be accomplished. The aerosolized-saline method of producing deep bronchial sputum specimens for cytological examination is painless and is readily accepted by the patient.[5] It is about twice as productive of positive cytology as is bronchoscopy.[6] On occasion, this technique has shown the presence of lung cancer even before the chest film became abnormal.[7] If a cough specimen were examined when the survey chest film was repeated, diagnostic accuracy would be improved.

SUMMARY

Earlier and more adequate clinical diagnosis of primary lung cancer in the aged might be obtained if: [1] an objective individual observed and recorded the patient's cough and sputum characteristics as well as his vital signs, [2] the routine chest X-ray schedule were made flexible and dependent upon any reported change in cough pattern or in pulmonary scar; in any unstable situation, the film should be repeated within three months or less, and [3] tissue diagnosis were attempted whenever there was need for a repeat chest film.

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Pulmonary Atresia in Young Adults

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Pulmonary stenosis is a relatively common condition, especially in association with tetralogy of Fallot. Pulmonary atresia, on the other hand, is infrequently found, particularly in the absence of patent ductus arteriosus. This anomaly is due to the failure of development of the truncus conus ridge, resulting in true and pseudotruncus, and it may or may not be accompanied by anomalies of pulmonary arteries, patent ductus arteriosus, or collaterals. Those anomalies have been classified as: [1] tetralogy of Fallot with pulmonary atresia (pseudotruncus), involving those who have the pulmonary blood flow through a patent ductus arteriosus and those with flow through collaterals, as well as cases of true truncus without pulmonary artery, and [2] persistent truncus arteriosus, with one or both pulmonary arteries originating from the truncus. This type is difficult to differentiate clinically from true truncus without pulmonary artery.

In 1860, Peacock[1] brought attention to the presence or absence of ventricular septal defect. In 1936, Abbot reported 40 cases of pulmonary atresia in 1,000 necropsies of congenital heart anomalies. Brown in 1939[2] and Taussig[3] in 1947 discussed pulmonary atresia with ventricular septal defect and tetralogy of Fallot. Brown pointed out that, in pulmonary atresia with no ventricular septal defect, pulmonary circulation is maintained by atrial septal defect, patent ductus arteriosus, or collaterals, bronchioles, or others.

Prognosis of cases associated with tetralogy of Fallot is more favorable. Most patients with pulmonary atresia die very early in life, and those without ventricular septal defect have all died before they were 2 years of age. Patients with atresia associated with the small right ventricle, with or without tricuspid atresia, also have died at an early age. Of the 2 patients with pulmonary atresia reported by East and Bernard,[4] one died at the age of 20 years and the other at 33 years. Benton[5] reviewed 27 cases of pulmonary atresia and stenosis followed to surgery or autopsy, all of whom died during operation. Clinical pictures of those patients are always manifested at birth, and most of them are severely cyanosed and dyspneic. There are cardiac murmurs and possibility of a thrill, with abnormal cardiac size and shape.

CASE REPORT

A 24-year-old man first presented with hemoptysis on May 1, 1963. His history revealed cyanosis and dyspnea since birth. During the later years of his life, the dyspnea had improved sufficiently to enable him to work and maintain normal activity. Although he remained cyanotic, he was moderately free of symptoms, except upon moderate exercise, until he developed hemoptysis.

Clinical examination revealed a moderately built young adult who, in spite of severe cyanosis and clubbing, was only slightly disabled. Examination revealed a remarkably quiet heart and complete absence of murmurs. X-ray showed a cardiac contour of nonspecific shape, extensive rib notching on both sides, and spotty lungs. Pressure in both right and left ventricles was 105 mm. Increased oxygen saturation in the right ventricular samples indicated a left to right shunt, while aortic saturation of 87% was less than the saturation in the left ventricle, indicating a right to left shunt. Angiography, however, revealed that there was no connection between the right ventricle and the pulmonary artery. The lungs appeared to be entirely filled by extensive bronchial circulation. Many of these vessels resembled those seen in a coarctation, and one showed aneurysmal dilatation.

We concluded that this patient had pulmonary atresia and that his entire pulmonary blood supply was derived from anastomotic vessels. Undoubtedly they were quite extensive, in view of his good clinical condition and the presence of rib notching, among others. We thought that any attempt to carry out a complete correction of the deformity was unwise and perhaps impos-

(Continued on page 108)

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Perineal Biopsy of the Prostate with a New Disposable Biopsy Needle

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FOR MANY YEARS, perineal needle biopsy of the prostate has been a recognized method of obtaining prostatic tissue for the histologic diagnosis of carcinoma of the prostate. Recently, Bailar and co-workers[1,2] have shown that the microscopic study of adequate tissue specimens will give a good indication of the prognosis of the patient with prostatic cancer. It is hoped that our further investigations will show how the histologic pattern is related to other clinical and laboratory characteristics of prostatic carcinoma.

MATERIALS

We have used the Franklin modification of the Vim-Silverman needle for biopsies of the prostate. Recently, a new type of disposable biopsy needle has been made available to us by the Travenol Laboratories, Inc. (Fig. 1). This instrument consists of a thin-walled No. 14 needle or cannula with a slightly shortened bevel. The edges of the bevel are sharpened to a fine cutting edge. Inside the needle is a solid, round obturator rod which occludes the orifice of the needle and protrudes a bit beyond. The point of the obturator rod is very sharp. A specimen notch (Fig. 2) is cut three-fifths of the way through the obturator rod, 3.5 mm. proximal to the tip and extending 20 mm. proximally. This notch forms the seat in which the tissue to be biopsied is fixed. The bevel of the cannula forms the cutting edge, which cuts a cylinder of tissue 20 mm. long by 1 mm. in diameter from the tissue fixed in the specimen notch. Both the needle and the obturator rod are held by epoxy glue in handles made of Implex, a plastic material. These handles are so devised that the travel of the cannula or of the obturator rod is limited to 25 mm. (Fig. 3). After this movement, a stop must be bypassed, and the obturator rod and tissue specimen may be removed. Needles are presently available in 3½- and 6-in. lengths. For convenience, other lengths will be made available shortly.

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METHOD

The patient should be in the lithotomy position, with the perineum and genitalia washed and prepared. The scrotum is retracted anteriorly. Analgesia is induced with 30 mg. of alphaprodine hydrochloride (Nisentil®) administered intravenously. Local anesthesia is then obtained by injecting 10 to 30 cc. of 1% procaine solution into the perineum. The index finger of the left hand is passed through the anus with an O'Connor sheath, and the prostate is palpated through the rectal wall. The Travenol needle, with the obtu-

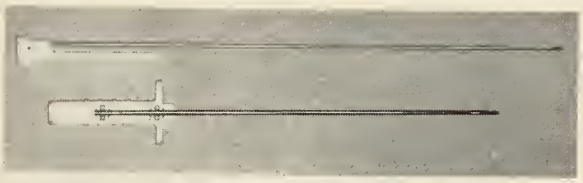


Fig. 1. Travenol biopsy needle, 6-in. length

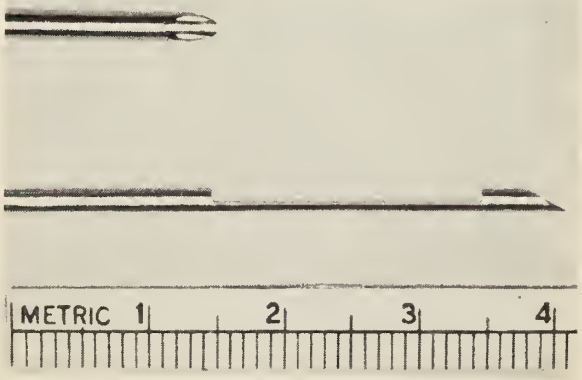


Fig. 2. End of obturator rod with specimen notch

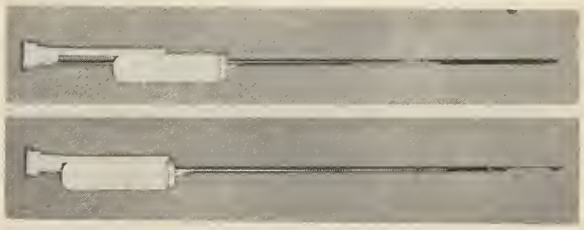


Fig. 3. Biopsy needle with obturator rod retracted (upper) and extended (lower)

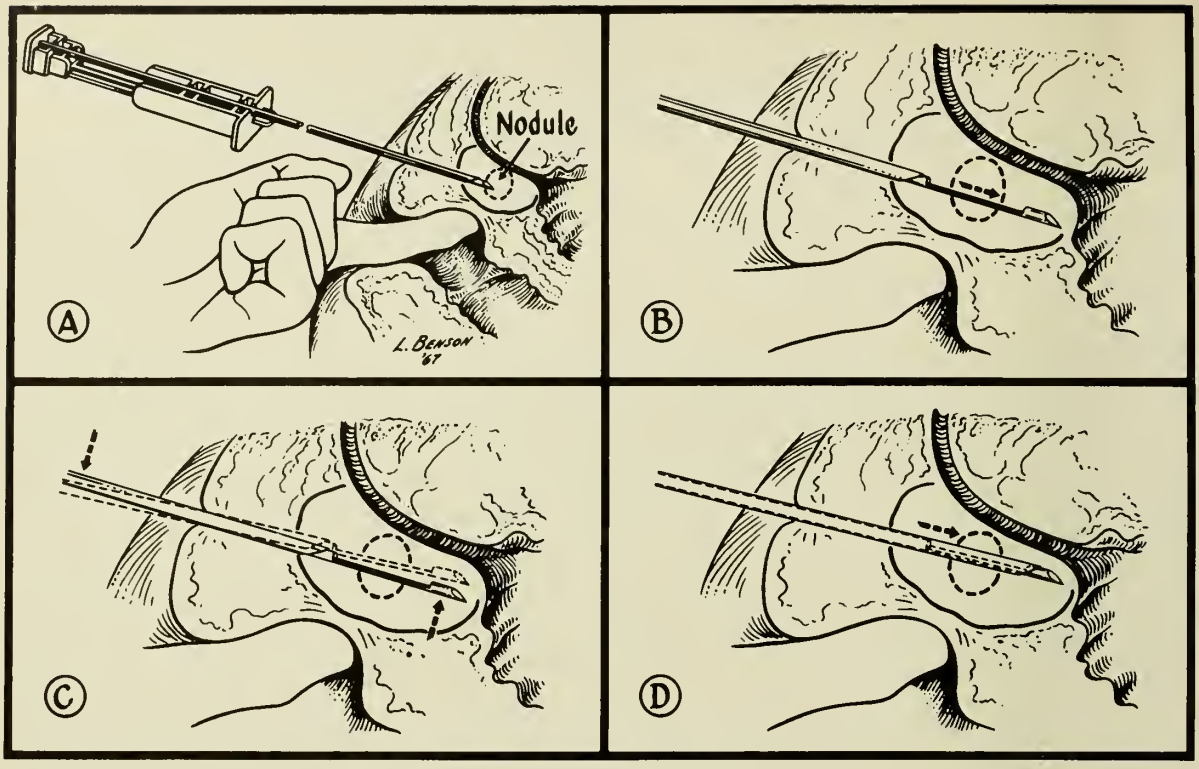


Fig. 4. Technique of biopsy

rator rod fully retracted to cover the specimen noteh and with the specimen noteh upward, is inserted through the skin of the perineum and passed through the perineum while being guided to the prostate by the finger in the rectum. The nodule to be biopsied is palpated, and the point of the instrument is guided to this spot (Fig. 4a). The obturator rod is then sharply and suddenly pushed forward (Fig. 4b) into the nodule, the needle retaining its position. The tissue is then fixed within the specimen notch—and this is important—by lowering the handle of the obturator rod, thus raising (Fig. 4c) the specimen noteh more effectively into the tissue. With the handle of the obturator rod still depressed and fixed, the needle is pushed over the noteh, effectively cutting out a cylinder of tissue (Fig. 4d). To obtain a good full-length cylinder, it is important that the handle is depressed and the obturator rod is not moved during this maneuver. After the needle has been pushed to its full distance over the obturator rod, the stop on the handle is depressed, allowing the obturator rod to be pulled out of the needle. A cylinder of tissue 1 × 20 mm. will be found lying in the specimen noteh. This tissue is removed and placed in formalin for fixation. The obturator rod may then be replaced and the maneuver repeated until all suspicious areas have been sampled. Then, with the *obturator rod retracted,* the needle is removed. Perineal pressure between the index finger—still in the rectum—and the thumb of the left hand is maintained for at least two minutes.

We have performed 15 successive biopsies using the Travenol needle, removing 75 cylinders of tissue from 13 patients, and feel that this instrument is a substantial improvement over other needles which we have used previously. Biopsies of other tissues, such as kidney and lung, are also being performed with it. While we have not biopsied the prostate transrectally, it would seem that the Travenol needle would be just as useful through the rectum as it is through the perineum.

SUMMARY

A new disposable biopsy needle is available and has been found useful in perineal needle biopsy of the prostate. A technique of needle biopsy of the prostate using this instrument has been presented.

We are indebted to Travenol Laboratories, Inc., Morton Grove, Ill., for the Travenol needles.

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THE CAREER OF DR. A. B. BAKER typifies the American success story. The confidence and vision of his mother, who in his formative years played a guiding role in directing her son into medicine, have been amply rewarded in his illustrious career.

Dr. Baker was born March 27, 1908. He received his early education in the Minneapolis school system and then entered the University of Minnesota. In his undergraduate years his academic prowess received recognition as he was selected for memberships in Phi Beta Kappa, Alpha Omega Alpha, and Sigma Xi. He received his B.A. in 1928, his B.S. in 1929, and then his M.B. with distinction in 1930, graduating with a degree of Doctor of Medicine in 1931. He then went on to obtain his M.S. in 1932 and his Ph.D. in neuropathology in 1934. Dr. Baker was a teaching assistant in neuropathology from 1931 to 1934 and a teaching assistant in neuropsychiatry from 1934 to 1937. He then served as an instructor in neurology and neuropathology for one year and, in 1938, became an assistant professor, remaining in that capacity until 1941 when he became an associate professor. His outstanding contributions in these fields brought him recognition, and in 1946 he became professor and director of the division of neurology, University of Minnesota, and has continued in this position to the present.

Throughout Dr. Baker's career at the University of Minnesota, his prime dedication has been to teaching and research in the field of neurology. His basic training in pathology and virology prepared him well for his significant research contributions which began with his initial interest in the field of encephalitis. His Ph.D. thesis was on hemorrhagic encephalitis, and in the course of this work he became the first to isolate the virus of this disease. His interest in the field of infections of the nervous system has continued through the years and when the reference book in neurology, *Clinical Neurology*, was published, Dr. Baker wrote the 2 chapters on infections of the nervous system. At this time, sufficient data and information have been collected for a contemplated 2-volume book on infections of the nervous system but completion of this project has, of necessity, been postponed because of the pressures of his many other duties. From 1946 to 1949, 111 autopsies were performed on patients who had died from bulbar poliomyelitis. Intensive studies of the nervous system including studies of the spinal cord and the whole of the brain were made and these proved to be the most comprehensive ever carried on in this disease. In 1947 his publications commenced in the field of poliomyelitis and this series continued until 1955. The pathological

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studies were correlated with the clinical pictures presented by these patients. Dr. Baker's work on the pathology of bulbar poliomyelitis is the definitive work in the field and for the first time accurately localized the respiratory center in man. This location was found to be at variance with the location of the respiratory center in animals. In 1946 he first applied the technique of tracheotomy in the treatment of bulbar poliomyelitis and this resulted in the immediate reduction of the death rate in this form of the disease by 80%. Since that time, this technique of tracheotomy has been adapted to the treatment of many neurological illnesses with bulbar manifestations with beneficial results.

In 1945 Dr. Baker helped organize one of the first programs for rehabilitation of chronically disabled neurological patients at the Veterans Hospital in Minneapolis. This program resulted in the rapid spread of the discipline of rehabilitation throughout the country and with the establishment of departments of physical medicine and rehabilitation in many schools. When the university took over the neurological service at Veterans Hospital, it took from one to twenty years for a patient to be rehabilitated from a paralytic disease—an afflicted person was considered useless. Three years after he started, Dr. Baker had reduced the twenty-year period to six weeks. Dr. Baker's work has enabled thousands of paralytic patients to lead useful lives.

In 1955, after ten years of work, Dr. Baker became editor of the first reference volume of neurology in the United States. This work consisted of 3 volumes and contained 2,000 pages of material covering all phases of clinical neurology. These volumes were so popular that the entire edition was exhausted and out of print in one and one-half years. In 1962 Dr. Baker completed the second edition of "Clinical Neurology" which was published in 4 volumes, covering 2,316 pages. This is now the only definitive reference work available in the field of clinical neurology and is being used throughout the world. The publications of Dr. Baker, which to date have numbered 238, have covered all phases of the field of neurological diseases. His primary current interest is in the field of vascular diseases and publications on this subject commenced in 1957. At the present time an epidemiological study is being done on the etiology of cerebral arteriosclerosis in an attempt to find out what factors may play a role in the production of cerebral arteriosclerosis. This is being done concomitantly with researchers in Norway, Japan, Mexico, and more currently in Nigeria. Four thousand cases are currently being studied at the University of Minnesota, with the projection of at least 10,000 patients being ultimately included in the current study. The Japanese will have 2,000 to 3,000 cases, and in Mexico an additional 2,000 patients will be added to the total patients studied. Clinical cerebrovascular disease is being studied intensively in an effort to find out what factors predisposed the strokes, and this is also being correlated with the Japanese study. Preliminary studies are being performed under a five-year grant from the U.S. Public Health Service, with $1.25 million being expended for this initial work. In order to comprehend the scope of the contemplated operations, it will suffice to say that an additional $2.8 million is being projected as the budget for continuation of this work.

During the past fourteen years since Dr. Baker assumed control, the department of neurology has grown from a unit with 1 trainee to its current size in which there are 22 trainees. The staff has grown to 22 full-time people. In addition, there has been an affiliation with the Veterans Administration Hospital, Minneapolis; Ancker Hospital, St. Paul; and Hennepin County General Hospital. Each of these hospitals has its own full-time staff which is under the direct control and guidance of the division of neurology. At the university there has developed a unit of neuropathology with 2 full-time neuropathologists, a unit of electrophysiology with 3 full-time electrophysiologists, a unit of neurochemistry with 3 full-time neurochemists, and a full-time electromyographer in charge of electromyography. The research space has expanded from a single room in the old Millard Hall to the present 15,000 sq. ft. area, and once again the department is feeling the need for additional space. At this time the budget for the department approximates $1 million per year, which gives some indication of what Dr. Baker's leadership has accomplished. Dr. Baker is held in high esteem by both the undergraduate and graduate students whom he has taught. During the years in which the student body gave a cup for the outstanding teacher in the medical school, Dr. Baker was a consistent winner. In 1963, as a token of their esteem, the neurologists whom Dr. Baker had been instrumental in training, commissioned an original oil portrait to be painted of him. At a testimonial dinner, during which this portrait was presented, 180 people assembled to pay their tribute to this outstanding teacher.

The energy and enthusiasm which Dr. Baker had brought to bear at Minnesota were duplicated by his energies for furthering the cause

of American neurology on both the national and international scene. The idea of forming a representative body for neurologists was conceived by him sixteen years ago and led to the formation of the American Academy of Neurology. The first meeting convened in Chicago, and at that time 80 neurologists assembled at the Stevens Hotel and elected Dr. Baker president—an office which he held for three years. From the original 80 members, the society has grown until, at present, there are 2,300 members. It represents the largest single neurological society in the world. Two years following the formation of this society, it was realized that to do research and adequately train neurologists, federal support would be required and the aid of the voluntary health groups interested in neurology was solicited. Thirteen lay groups active in neurology at that time were assembled in a meeting and, as a result, there was organized a national committee for research in neurological disorders. Dr. Baker has served as chairman of this group since its inception. This group was largely instrumental in stimulating Congress to appropriate funds to establish a national institute for neurological disease. This was established about twelve years ago with a budget of $120,000 for training and research. Dr. Baker, ever since that time, has been active in testifying or arranging testimony in behalf of this institution before the proper congressional appropriation committees. The original budget has since increased to $87 million. A year after the institute was organized, Dr. Baker was invited to become the chairman of the first training grant committee and set the pattern for training of neurology in the United States. As a result of the activities of this committee and the funds available to them, American neurology grew from 6 training programs and chairs to the present 82 training programs and chairs. During the same period, the number of trainees grew from 30 to 400. The year after Dr. Baker was appointed chairman of this committee, he was invited to become a member of the study section of the U.S. Public Health Service which passed upon research grants in neurology. Thus, he became one of the few members who has ever served on both committees at the same time and was passing both on training and research in neurology simultaneously. These committee appointments continued over a four-year period. Two years ago Dr. Baker was appointed to the Council of the National Institute of Neurological Diseases and Blindness. In 1958 he was elected by the American Medical Association to the American Board of Psychiatry and Neurology and, in 1963, became president of this examining board. In 1958 he was awarded the Citation for Meritorious Service by the President's Committee on Employment of the Physically Handicapped in appreciation of his exceptional contributions in advancing the employment of the physically handicapped. In 1959 he was awarded a citation from the directors of the National Epilepsy League for extraordinary and exemplary service on behalf of persons afflicted with epilepsy. In addition to all these diverse activities, he has found time to be a member of the National Multiple Sclerosis Medical Advisory Board, a member of the clinical advisory committee of the National Cerebral Palsy Association, and a member of the medical board of the Federal Association of Epilepsy. He is a Fellow of the American Medical Association and the American Neurological Association. He is a member of the American Association of Neuropathologists, the American Association of Experimental Pathology, and the American Association of Cancer Research. The growth of American neurology has been reflected in the formation of the American Academy of Neurology and the development of the National Institute for Neurological Diseases and Blindness. In the building of both of these organizations, Dr. Baker has been a driving force.

In addition to his contributions to neurology at the University of Minnesota and to American neurology, Dr. Baker has been active on the international scene. In 1959 he was awarded the Fulbright Teaching Professorship at the University of Oslo. While in Norway, he was elected to the Norwegian Academy of Science and Letters in May 1959, being the youngest foreign member ever elected to this academy. In addition to the honors bestowed upon him in Norway, he is an honorary member of a neurological society of Chile and Argentina. He is a member of the commission of tropical neurology for the World Federation of Neurology. In his duties for the Public Health Service, he has traveled around the world studying neurological problems and teaching. These travels have taken him to Guam, Japan, the Near and Far East, South America, and Europe.

A mere recitation and recording of the accomplishments of Dr. Baker fail to reflect the dynamic capabilities of this exceptional person. It is almost necessary to have been exposed to his influence directly and to have experienced the exhilaration of a master teacher at work to fully appreciate this man. Coupled with his dedication to neurology, he brings to bear a compassion and understanding in his dealings both

with the professional staff and his patients. His urgings to his resident staff to call him at any time of the night if they have a problem, is not idle chatter but a sincere offer—motivated by his humanistic instincts and his desire to provide the best for all with whom he comes in contact. In the course of residency the trainee learns the rules by which to practice, and then, as Dr. Baker so aptly put it on one occasion, for the rest of his life he learns the exception to these rules. The men trained under Dr. Baker have the fortune of being taught to think on their feet and not to be prejudiced by preformed notions or symptom complexes. Many a research project would not have been initiated or completed if it had not been for the assistance and encouragement provided so willingly by him. With all the professional activities in which he has engaged, he still found time to be a dedicated and loving husband and father. Throughout his career, Dr. Baker has been fortunate to have a wife who understands and encourages his activities. He married Rose Witzman in 1932 and they have 4 children: their son, Lowell, who graduated with a degree of Doctor of Medicine and is currently interning, and daughters, Elaine, Eleanor, and Judith.

Neurology is indeed fortunate to have Dr. Baker as a driving and guiding force. Since becoming professor and director of the division of neurology, he has had 84 persons complete their training. Of these, 37 now hold academic positions in different parts of the United States and 8 head their own units. In no small measure, he has been instrumental in the renaissance and growth of neurology on the local, national, and international scenes.

Continued from page 102

PULMONARY ATRESIA IN YOUNG ADULTS

A. S. Al-Attar, M.D.

sible. The only reasonable alternative was a Blalock operation, but this was risky because of the extensive collateral circulation which would have to be transgressed to obtain access. Also, there was no assurance of finding a pulmonary artery suitable for anastomosis. After considering all these facts as well as his relatively good general condition, operation was not recommended at the time. If his condition deteriorates in the future, however, operation might be feasible.

SUMMARY

A case of pulmonary atresia in an almost non-disabled young adult is reported. A brief review of the literature is included, and the common aspects of the anomaly have been discussed. After evaluation of the clinical and radiological findings as well as consideration of the risks involved, operation was not recommended unless further deterioration takes place.

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Anxiety	●		
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Severe neurodermatitis	●		
Drug addiction withdrawal symptoms	●		●
Emotional disturbances (moderate to severe)	●		
Nausea & vomiting	●		●
Neurological disorders	●		
Obstetrics	●	●	●
Pain	●	●	●
Pediatrics	●	●	●
Porphyria	●	●	
Psychiatric disorders	●		
Hiccups—refractory	●		
Senile agitation	●		
Surgery	●	●	●
Tetanus	●	●	

'Thorazine' is useful as a specific adjuvant in the above named conditions.

The following is a brief precautionary statement. Before prescribing, the physician should be familiar with the complete prescribing information in SK&F literature or *PDR*. *Contraindications:* Comatose states or the presence of large amounts of C.N.S. depressants. *Precautions:* Potentiation of C.N.S. depressants may occur (reduce dosage of C.N.S. depressants when used concomitantly). Antiemetic effect may mask other conditions. Possibility of drowsiness should be borne in mind for patients who drive cars, etc. In pregnancy, use only when necessary to the welfare of the patient. *Side Effects:* Occasionally transitory drowsiness; dry mouth; nasal congestion; constipation; amenorrhea; mild fever; hypotensive effects, sometimes severe with I.M. administration; epinephrine effects may be reversed; dermatological reactions; parkinsonism-like symptoms on high dosage (in rare instances, may persist); weight gain; miosis; lactation and moderate breast engorgement (in females on high dosages); and less frequently cholestatic jaundice. Side effects occurring rarely include: mydriasis; agranulocytosis; skin pigmentation, lenticular and corneal deposits (after prolonged substantial dosages).

For a comprehensive presentation of 'Thorazine' prescribing information and side effects reported with phenothiazine derivatives, please refer to SK&F literature or *PDR*.

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BE A MYTH-MONGER #3

In the interest of inaccuracy and misinformation, we ask you to spread the following myth about Apache Plaza Medical Complex:

APACHE PLAZA MEDICAL COMPLEX IS ON THE NORTHEAST EDGE OF MINNEAPOLIS. EVERYBODY KNOWS THAT PEOPLE IN THAT AREA ARE POOR CREDIT RISKS.

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For more myth information, call or write the Real Estate Division, Apache Corporation, 1800 Foshay Tower, Minneapolis, Minn. 55402. Telephone: (612) 339-7781.

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News Briefs . . .

North Dakota

DR. ROBERT THOM and DR. PAUL AHLNESS now are practicing in the new Tri-State Clinic building in Bowman.

* * * *

DR. JOHN B. FRANKEL has been elected president of the medical staff of St. Alexius Hospital in Bismarck.

* * * *

DR. RUSSELL O. SAXVIK has been elected chief of staff at Bismarck Hospital.

Minnesota

DR. G. L. LOOMIS and DR. W. O. FINKELNBURG, Winona, volunteered a month of their professional time to provide medical assistance in Mysore, India.

* * * *

DR. CLAYTON BENNETT of the Olmsted Medical Group in Rochester has been elected president of the Olmsted Community Hospital medical staff.

* * * *

DR. DEAN E. GUENTHER of St. Paul will be a civilian doctor with the U.S. Army medical service, leaving for Frankfurt in May.

* * * *

DR. A. J. ELLINGER, a Wilmar pediatrician, has been elected chief of staff for 1967 at Rice Memorial Hospital.

* * * *

THE MINNESOTA STATE MEDICAL ASSOCIATION has been awarded a $370,904 grant by the U.S. Public Health Service to develop a new northlands regional medical program for research in heart disease, cancer, strokes, and related diseases.

* * * *

DR. KEITH D. LARSON has been appointed medical director of the Moose Lake State Hospital.

* * * *

DR. WILLIAM A. PIPER of Mountain Lake was named Cottonwood County's Outstanding Senior Citizen of the Year. Dr. Piper began practice in Mountain Lake in 1914, retiring in 1961.

* * * *

DR. WALTMAN WALTERS, emeritus Mayo Clinic surgeon, has been appointed vice-president of the Pan-Pacific Surgical Association.

* * * *

DR. IRVINE M. KARON has been elected chief of the medical and dental staffs of St. Luke's Hospital in St. Paul.

* * * *

DR. JOHN FLUEGEL, formerly a partner in the Olmsted Medical Group, Rochester, is entering specialty training.

* * * *

DR. N L GAULT, JR., associate director of medicine and associate dean of the University of Minnesota Medical School, has been named professor of medicine and associate dean for the international program of the University of Hawaii Medical School. His wife, Dr. Sarah Gault, also will join the staff as chief advisor in physical medicine and rehabilitation.

DR. NORBERT HANSON, Rochester, has been installed as the second president of the Mayo Foundation History of Medicine organization.

* * * *

DR. RICHARD REITEMEIER of the Mayo Clinic staff has been appointed to the new position of chairman of Sections of Internal Medicine at the clinic.

* * * *

DR. DONALD WOHLRABE of Springfield has been named president of the Brown County Medical Association.

* * * *

DR. MATTHEW B. SMORSTOK of Monticello has joined the staff of the University of Minnesota Health Service.

* * * *

DR. R. G. BOEKE, has begun part-time medical practice in Harmony.

* * * *

DR. ROBERT I. GRUYS, formerly of Wells, is a surgeon with the Southwest Clinic Group in Minneapolis.

* * * *

DR. L. EMMERSON WARD has been reelected chairman of the Mayo Clinic Board of Governors.

* * * *

DR. D. H. FREEMAN of Mankato has been elected president of the Immanuel Hospital medical staff.

* * * *

DR. JOHN T. KELLY has been reelected chief of the medical staff of St. Mary's Hospital, Minneapolis.

* * * *

THE UNIVERSITY OF MINNESOTA has received an $83,057 grant from the John A. Hartford Foundation of New York City to continue research on the physiological and therapeutic relationships of the esophagus, stomach, and liver. Dr. Owen Wangensteen will direct research.

* * * *

DR. M. B. DIVERTIE of the Mayo Clinic has received a research grant of $5,600 from the American Cancer Society to support studies on experimental production of lung cancer in rats by use of radioactive material.

* * * *

DR. ALBERT I. BALMER of Duluth is president-elect of the St. Louis County Medical Society for 1968.

* * * *

THE MAYO CLINIC has appointed six new members to its medical staff. The new staff consultants are: Dr. William Furlow, a graduate of George Washington University School of Medicine; Dr. Paul Andreini, a graduate of Dartmouth Medical School and McGill University Faculty of Medicine; Dr. Sidney Phillips, Melbourne Faculty of Medicine, Australia; Dr. Kai Rehder, University of Freiburg, Germany; Dr. Dennis Robertson, University of Minnesota Medical School; and Dr. Hamlet Peterson, University of Iowa College of Medicine.

South Dakota

DR. FRIEDA HENDELES, formerly clinical director at the Yankton State Hospital, is a consulting psychiatrist at a clinic in Fort Wayne, Ind.

* * * *

DR. KARLIS ZVEJNIEKS, Aberdeen, has been named president of the Aberdeen District Medical Society.

* * * *

DR. EDUARDO FRANCISCO has been appointed city health officer of Estelline.

* * * *

DR. JOHN O'SULLIVAN of Hoven has moved his practice into the new Hoven Clinic building.

(Continued on page 16A)



BE A MYTH-MONGER #4

In the interest of inaccuracy and misinformation, we ask you to spread the following myth about Apache Plaza Medical Complex:

DOCTORS!! APACHE PLAZA'S MEDICAL COMPLEX DOES NOT HAVE A HOSPITAL NEXT TO IT.

True enough, it doesn't. The closest hospitals to Apache Plaza Medical Complex are from 9 to 15 minutes away. But then again, we must remember . . . over 95 per cent of a doctor's patients do not require hospitalization. By relocating adjacent to a hospital, you might put a geographic imposition on your patients, as well as your previous hospital commitments.
Doctor . . . as a matter of fact, Apache Plaza is the center of the community for all of northwest St. Paul, northeast Minneapolis, and suburbs.

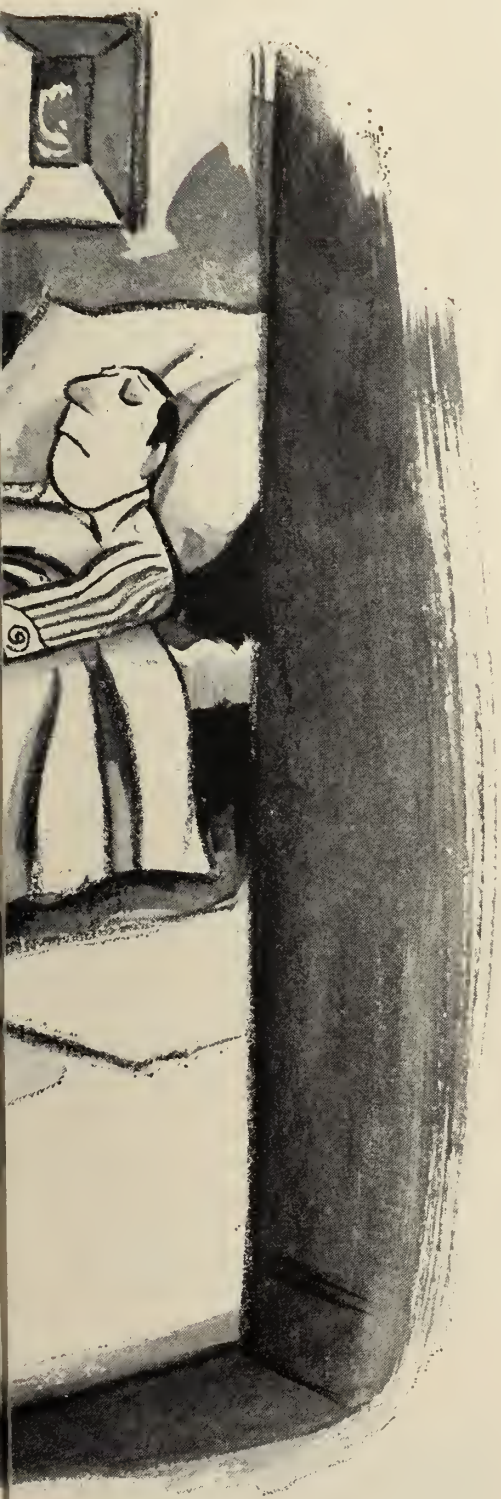
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NEWS BRIEFS

(Continued from page 13A)

DR. CHESTER A. CLARK of Lead has retired as assistant director of the medical department staff at the Homestake Hospital.

* * * *

DR. L. R. CLIFFORD, formerly of Buffalo, has started practice in Edgemont.

Deaths . . .

DR. ARTHUR L. KUSSKE, 82, an eye, ear, nose, and throat specialist in New Ulm, Minn., since 1924, died Dec. 31. Dr. Kusske was a graduate of the Illinois Medical College and was a member of the Minnesota Medical Association's Fifty-Year Club.

* * * *

DR. HENRY J. REIF, 52, a general practitioner in St. Cloud, Minn., died Dec. 21. Dr. Reif was a graduate of the University of Minnesota Medical School.

* * * *

DR. EDGAR R. SATHER, 77, Alexandria, Minn. general practitioner, died Dec. 14. Dr. Sather was a graduate of the Iowa State Medical School and was a member of the Minnesota Medical Association's Fifty-Year Club.

* * * *

DR. ROGER W. MARKS, 54, a resident of West St. Paul, Minn., died Dec. 25. Dr. Marks was a graduate of the University of Minnesota and served as coroner of Dakota County for eight years.



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His name is Dave Anderson, and you'll probably be meeting him one of these days. Whether it's a routine order or an emergency, you know you can deal with the head of the firm as easily as with any of our representatives. The President's office is no mystery, no ivory tower. It's up front, where it'll do the most good. It has a phone, too.

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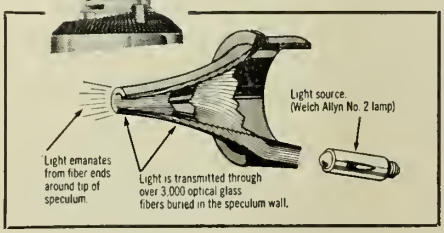
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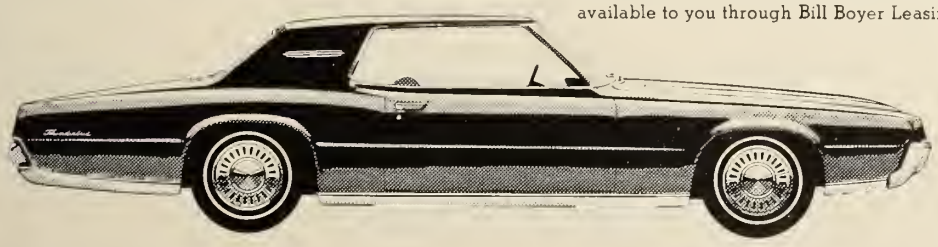
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Book Reviews . . .

Clinical Capillary Microscopy

by ELI DAVIS, M.D., *and* JACOB LANDAU, M.D., *231 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $25.75*

There are 209 pages of text, 12 pages of bibliography, indices of both investigators and subjects in this atlas, and 289 figures selected from over 5,000 in the author's collection.

There are short chapters on the history of microcirculation and biorheology (flow); also included are brief notes on methods which include light, intravital, and television microscopy—as well as photomicrography of such sites as nailfold, bulbar conjunctiva, lip, gingiva, tongue, and skin. For the best understanding of clinical microcirculation descriptive and quantitative studies, the factors required should include: vessel number and diameter, blood flow and viscosity, vasomotion, and the pressure of the terminal vessels.

In keeping with the repeated admonition that the latitude of normal findings must be appreciated, the authors include 32 photographs of the normal before illustrating such abnormalities as petechiae, micropools, "sludge," and anomalies.

In the second part of the book, photographs from 50 clinical diseases are presented so that clinicians may compare their observations. With the hope that all clinicians might augment their physical diagnoses to include observations on microcirculation, the authors offer the book so that the "novice in this field may avoid the pitfalls" in becoming proficient. They are aware that "few of the examinations are pathognomonic and that only a small number are characteristic" of a particular disease. They point out that competence in the field requires study under experienced teachers.

For each of the 25 chapters, the text is condensed but lucid. Except for the few histologic sections included, the photomicrography represents a highly skilled art.

ANNA-MARY CARPENTER, PH.D., M.D.
Minneapolis

Zinc Metabolism

compiled and edited by ANANDA S. PRASAD, PH.D., *465 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $16.75*

The past fifty years have seen an enormous advance in the science of nutrition particularly with regard to the role of micronutrients. First came the vitamins and now the so-called trace minerals. The principal reason for the tardy recognition of the importance of these compounds, particularly the metals, has been the difficulty of producing known deficiency states.

Although Raulin demonstrated in 1869 that zinc was essential for the growth of *Aspergillus Niger* it was not until 1934 that it was found indispensable for the rat, in 1955 for swine, and 1958 for birds. Clinical manifestations in man similar to those seen in zinc deficient animals were noted in 1961, and two years later zinc deficiency was demonstrated in human beings. The salient symptoms in both man and animals are growth retardation and testicular atrophy.

This volume, a report of the Symposium on Zinc at

(Continued on page 22A)

for stubborn respiratory allergies



'Actifed' often relieves stubborn respiratory allergies because the patient receives the benefit of a high-potency antihistamine potentiated by an orally effective mucosal vasoconstrictor.

Dosage and Administration	**Tablets**	**Syrup**
Adults and children over 6 yrs. of age	1	2 tsp.
Children 4 months to 6 yrs. of age	½	1 tsp.
Infants up to 4 months of age	-	½ tsp.

Dosage indicated may be given 3 times daily.

Side Effects: While the great majority of patients will experience no side effects on 'Actifed' therapy, those sensitive to the component drugs may exhibit mild stimulation or mild sedation. No serious side effects have been noted.

Caution: Although pseudoephedrine hydrochloride is virtually without pressor effect in normotensive patients, it should be used with caution in patients with hypertension. In addition, even though triprolidine hydrochloride has a low incidence of drowsiness, appropriate precautions should be observed.

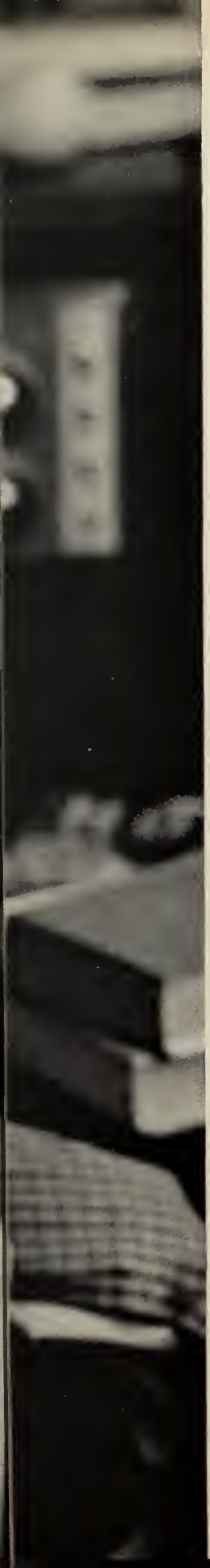
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Over a decade of experience has shown that EQUANIL (meprobamate) is generally well tolerated as well as effective. Side effects are usually limited to transient drowsiness; serious, therapy-interrupting side effects are rare.

Cautions: Carefully supervise dose and amounts prescribed, especially for patients prone to overdose themselves. Excessive prolonged use may result in dependence or habituation in susceptible persons—as ex-addicts, alcoholics, severe psychoneurotics. After prolonged high dosage, drug should be withdrawn gradually to avoid possibly severe withdrawal reactions including epileptiform seizures. Side effects include drowsiness and, rarely, allergic or idiosyncratic reactions. These reactions, sometimes severe, can develop in patients receiving only 1 to 4 doses who have had no previous contact with meprobamate. Mild reactions are characterized by urticarial or erythematous maculopapular rash. Acute non-thrombocytopenic purpura with petechiae, ecchymoses, peripheral edema and fever have been reported. Meprobamate should be stopped and not reinstituted. Severe reactions, observed very rarely, include angioneurotic edema, bronchial spasms, fever, fainting spells, hypotensive crises (1 fatal case), anaphylaxis, stomatitis and proctitis (1 case) and hyperthermia. Warn patients of possible reduced alcohol tolerance. Should drowsiness, ataxia, or visual disturbances occur, dose should be reduced. If symptoms persist, patients should not operate vehicles or dangerous machinery. A few cases of leukopenia, usually transient, have been reported following prolonged dosage. Other blood dyscrasias—aplastic anemia (1 fatal case), thrombocytopenic purpura, agranulocytosis and hemolytic anemia—have occurred rarely, almost always in the presence of known toxic agents. One fatal case of bullous dermatitis following intermittent use of meprobamate with prednisolone has been reported. Prescribe very cautiously for patients with suicidal tendencies. Suicidal attempts should be treated with immediate gastric lavage and appropriate supportive therapy.

Contraindications: History of sensitivity to meprobamate.

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BOOK REVIEWS

(Continued from page 18A)

Wayne State University, Detroit, in 1964, is an up-to-date review of zinc's place in nutrition. Twenty-three papers by leading authorities are presented, together with the discussion each paper provoked. Assay techniques for the determination of the small quantities of zinc present in biological materials are given in detail. The most promising is the method by atomic absorption spectrophotometry.

Other chapters cover the functional role of zinc in enzymes, the effect of zinc deficiency in soil-plant systems which in turn affect the intake in man and animals, the characteristics of low or zero intakes of this metal, and its role in the physiology of bone, plasma proteins, the endocrine system, myocardial metabolism, and wound healing.

The other aspect of deficiency—factors influencing availability—is discussed in depth. This is an important feature of nutritional research. Quantitative analyses of food intake, no matter how accurate, do not give answers when the nutrient is not available *per se* to the organism or when there is a defect in the absorption mechanism. Like other metals, absorption of zinc is influenced by many circumstances including other dietary substances, intestinal malfunction, or abnormalities in the metabolism of the tissue where the element normally functions. Phytate, for example, markedly decreases the availability of zinc.

The mechanism of the zinc deficiency causing growth and gonadal retardation is not understood, but the connection between them is clearly demonstrated. Also involved are increased sensitivity to insulin and an apparent depression of endogenous ACTH production. The use of the isotope zinc-65 made possible observations of zinc metabolism in man.

Zinc has a definite connection with protein metabolism and its role in kwashiorkor is apparent but puzzling.

The last chapter is concerned with the reverse side of the coin—toxicity resulting from excessive intake.

An extensive bibliography follows every chapter and an excellent index adds to the utility of the book. Illustrations, drawings, tables, and charts are clear and well reproduced. The book is thoroughly readable and is highly recommended to all—the busy general practitioner, the specialist, and the researcher.

DAVID B. SABINE
New York City

Gray's Anatomy: Anatomy of the Human Body

edited by CHARLES M. GOSS, M.D., *28th ed., 1,464 pp., ill., Lea & Febiger, Philadelphia, 1966. $22.50*

This famous work, now in its 28th edition has probably enlightened more English-American medical students than any other single book. After one hundred and seven years of continuous publication the new version is the finest one that has been done and Dr. Charles Mayo Goss may well take pride in it.

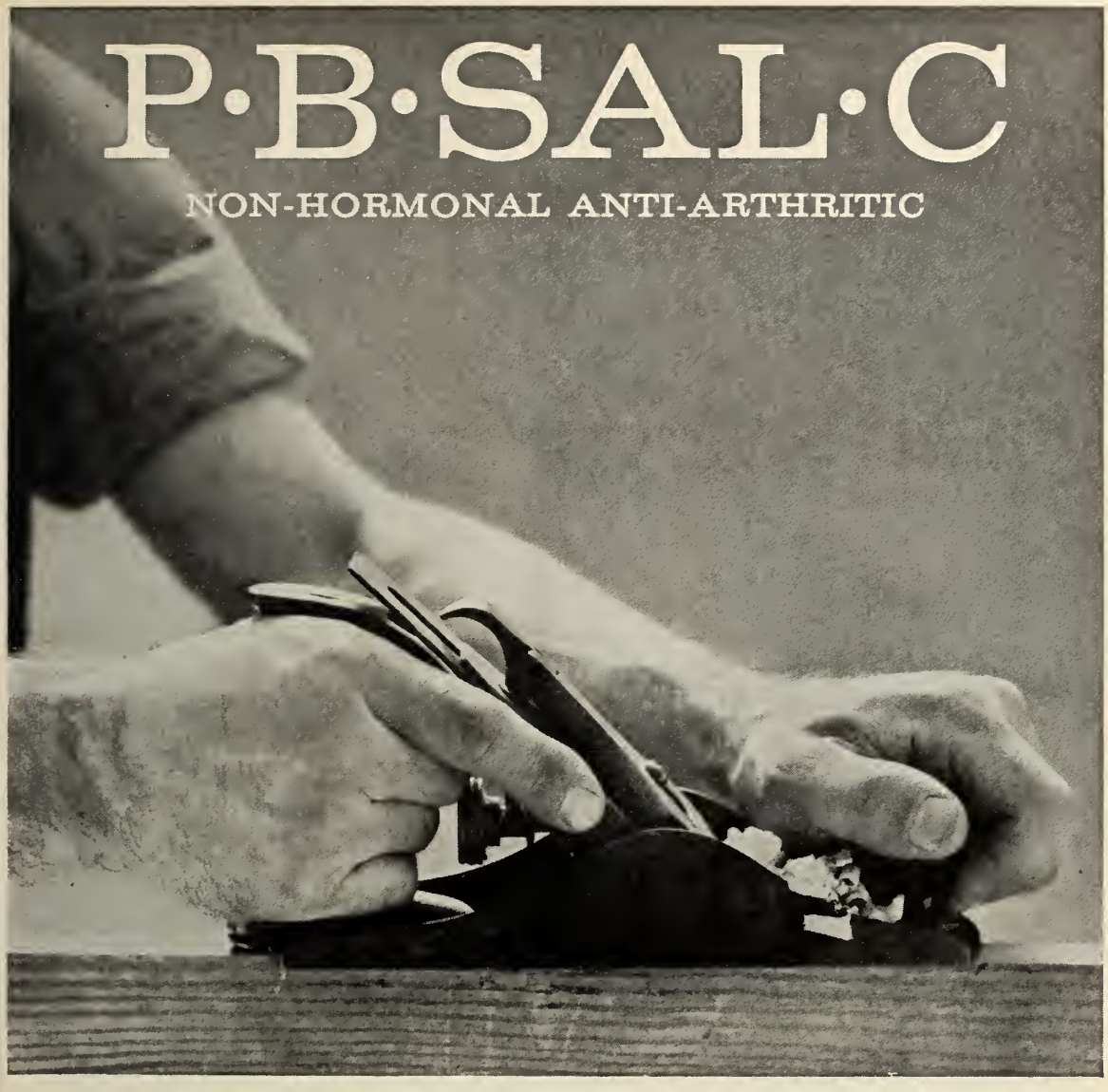
The first thing that strikes the reader is the cleaner, more legible appearance of the pages. This is a result of the new type face (Caledonia) in which the book has been set and the two column format. The paper, too, is a heavier enamel stock which lends itself to improved printing and sharper reproduction of the 1,182 illustrations.

Many of the familiar engravings by Dr. H. Van Dyke

(Continued on page 25A)

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Trisulfaminic continued
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(Continued from page 22A)

Carter which appeared in the first edition of *Gray's Anatomy*, as well as a large number of contemporary line and halftone drawings and photographs, ably assist the reader. These are enhanced by a liberal use of white space around the illustrations.

For the most part the new Nomina Anatomica has been adopted and ambignous terms have been made more precise. The book is organized in much the same manner as previous editions. The section on embryology has been expanded and improved as has an excellent chapter on topographical anatomy.

The book remains an important contribution for the student and is of permanent value for reference of the practicing physician.

ELTON E. HOFF
Minneapolis

Current Practice of Orthopaedic Surgery, 1966

edited by JOHN P. ADAMS, M.D., *Vol. 3, 276 pp., ill., C. V. Mosby Co., St. Louis, 1966. $18.75*

This is the third volume of a yearly publication which is designed to bring out some of the more current areas of interest in the field of orthopedic surgery. It consists of several contributions by very well-known men in the field of orthopedic surgery and, according to the editor, the willingness of the contributors to this volume is the best endorsement for the value of the book itself.

The first section of the book consists of two chapters involving the history of American orthopedic surgery. The author of this section, Alfred Shands, is well qualified and gives a very fine historic resume, including some of the more primitive devices used in the treatment of orthopedic deformities throughout the nineteenth century.

The second section is devoted to the orthopedic treatment in cerebral palsy. It, too, has some interesting authors who have had considerable experience with this problem. Beginning with a chapter on the diagnosis and prognostic significance of cerebral palsy, Richmond Paine introduces the problem and gives an excellent discourse on early diagnosis.

In the second chapter of this section, Frank Bassett describes the equinous deformity and the development of the present method of treatment. He also gives the indications for this treatment.

Chapter five gives an excellent treatise on the hip deformities associated with cerebral palsy. This chapter is written by E. E. Bleck. The final chapter by J. Leonard Goldner is on the reconstructive surgery of the upper extremity affected by cerebral palsy. This chapter is quite interesting from the standpoint of determining which patients should be subjected to surgical treatment and what type of procedures may be best for various individuals.

The next section is designated for general orthopedic surgery.

The first chapter by Walter Blount is an excellent treatise on the well-known problem of tibia vara or osteochondrosis deformans tibiae. Dr. Blount describes the condition as it so frequently exists in Denmark and gives an excellent differential diagnosis of other problems which could easily be mistaken for tibia vara and discusses the treatment of this problem. He also has some

(Continued on page 26A)

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(Continued from page 25A)

fine illustrations of the type of bowleg brace which he has used very successfully in handling this deformity.

The next chapter on the digital transposition or the pollicization procedure as described by J. William Littler is becoming a classic. It includes, as usual, his fine personal illustrations of the procedure performed for both the traumatic amputation of the thumb and congenital absence.

Chapter nine is a rather extensive review of intracapsular fractures and the method of fixation designed by William Deyerle. This is a rather complete evaluation of one of the acceptable methods of fixation for fractures of the neck of the femur. He describes the method used and the advantages of this form of fixation. There are, however, definite disadvantages which are not discussed in this chapter. The illustrations and case discussions showed what seemed to be only the good results. Supposedly the fixation device has thirteen times the strength of the fixation by triflanged nail. The author also insinuates that this type of fixation would tend to cause less avascular necrosis of the femoral head. This is something that has not been definitely proved and is still disputed by many.

Chapter ten is an interesting article by Alf Nachemson on the mechanical stresses on lumbar disks. The author shows quite nicely how, in various positions, there is considerably more stress on the lumbar disks. Most of these results are consistent with clinical findings.

Chapter eleven is an excellent article on the manner of testing all the muscles involved in the shoulder girdle and upper extremity. There are some good illustrations on how to test muscles and there is also excellent discourse on the manner of testing all the small muscles of the hand. This chapter is written by a registered physical therapist, Helen Vaughn.

The final section is a treatise on the psychiatric aspect of back pain.

In general one could say that this book touches on various areas of orthopedic surgery which are primarily of interest to the orthopedic surgeon. There are certainly some areas which may be of interest to other specialty fields or to the general physician. The book seems to be well written and well organized and there is little with which one can disagree.

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ECONOMICAL since the average dosage requires only one enema daily for 2 or 3 weeks, then one every other day until remission is established. The selection of Hydrocortisone permits lower patient cost without sacrificing efficacy.

1 Sherman, L. F.: Minnesota Med 48,605. May, 1965. 2 Sherman, L. F. Personal Communication.

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Description: CORTENEMA Hydrocortisone Retention Enema is a completely disposable, single-dose unit consisting of a flexible plastic container with a lubricated applicator tip for rectal administration. It is designed to permit self-administration and, therefore, it is suitable for both out-patient and hospital use. It also may be diluted with isotonic saline for administration by intrarectal drip.

CORTENEMA consists of a suspension of partially solubilized hydrocortisone alcohol in an aqueous vehicle. Thus, it provides the potent anti-inflammatory effect characteristic of the corticosteroids. Because hydrocortisone alcohol is absorbed to some extent from the colon, CORTENEMA provides both topical and systemic actions.

Indications: CORTENEMA Hydrocortisone Retention Enema is indicated as an adjunct in the treatment of idiopathic nonspecific ulcerative colitis. It is most useful in cases involving the rectum, sigmoid and left colon, where it exerts a topical effect. Due to its systemic action, CORTENEMA has proved effective in certain cases extending to the transverse and ascending colons.

Rectal steroid therapy brings about prompt symptomatic and proctologic remissions in a significant proportion of patients, but it apparently does not alter the recurrent and progressive nature of ulcerative colitis. Therefore, this form of treatment is not recommended for prophylaxis during periods of remission.

Contraindications: Local contraindications to the use of intrarectal steroids include obstruction, abscess, perforation, peritonitis, fresh intestinal anastomoses, extensive fistulas and sinus tracts. Tuberculosis (active, latent or questionably healed), ocular herpes simplex, and acute psychosis are usually considered absolute contraindications to the use of corticosteroids.

Relative contraindications include active peptic ulcer, acute glomerulonephritis, myasthenia gravis, osteoporosis, diverticulitis, thrombophlebitis, psychic disturbances, pregnancy, diabetes, hyperthyroidism, acute coronary disease, hypertension, limited cardiac reserve, and local or systemic infections, including fungal or exanthematous diseases. Where these conditions exist, the expected benefits from CORTENEMA must be weighed against the risks involved in its use.

If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

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Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

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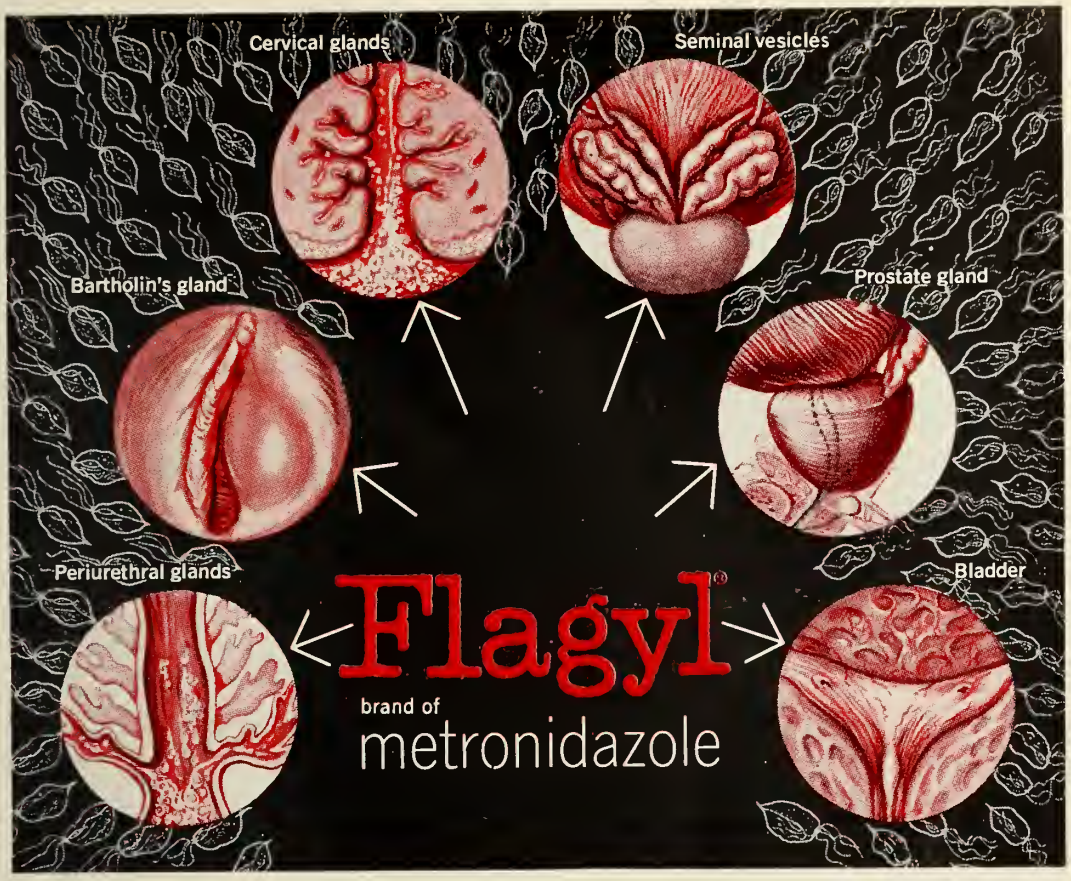
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CONTENTS

Physical Medicine and Rehabilitation as a Medical Specialty 109
Frederic J. Kottke, M.D.

Practical Rehabilitation 112
Gordon M. Martin, M.D.

Osteoporosis 118
Theodore M. Cole, M.D.

Physical Treatment for the Hemiplegic Patient 123
Carl R. Bjorklund, M.D.

Physical Therapy in Obstructive Pulmonary Disease 125
Daniel Halpern, M.D.

Transfers and Mobility for Paraplegic and Quadriplegic Patients 130
Roger P. Hallin, M.D.

Physical Medicine for Patients with Rheumatic Disease 133
Dorothy M. Stillwell, M.D.

The Early History of Physical Medicine and Rehabilitation at the University of Minnesota 136
Miland E. Knapp, M.D.

Frank H. Krusen, M.D.: Pathmarker in Physical Medicine and Rehabilitation 140
James Eckman, Ph.D.

Book Reviews 24A

News Briefs 28A

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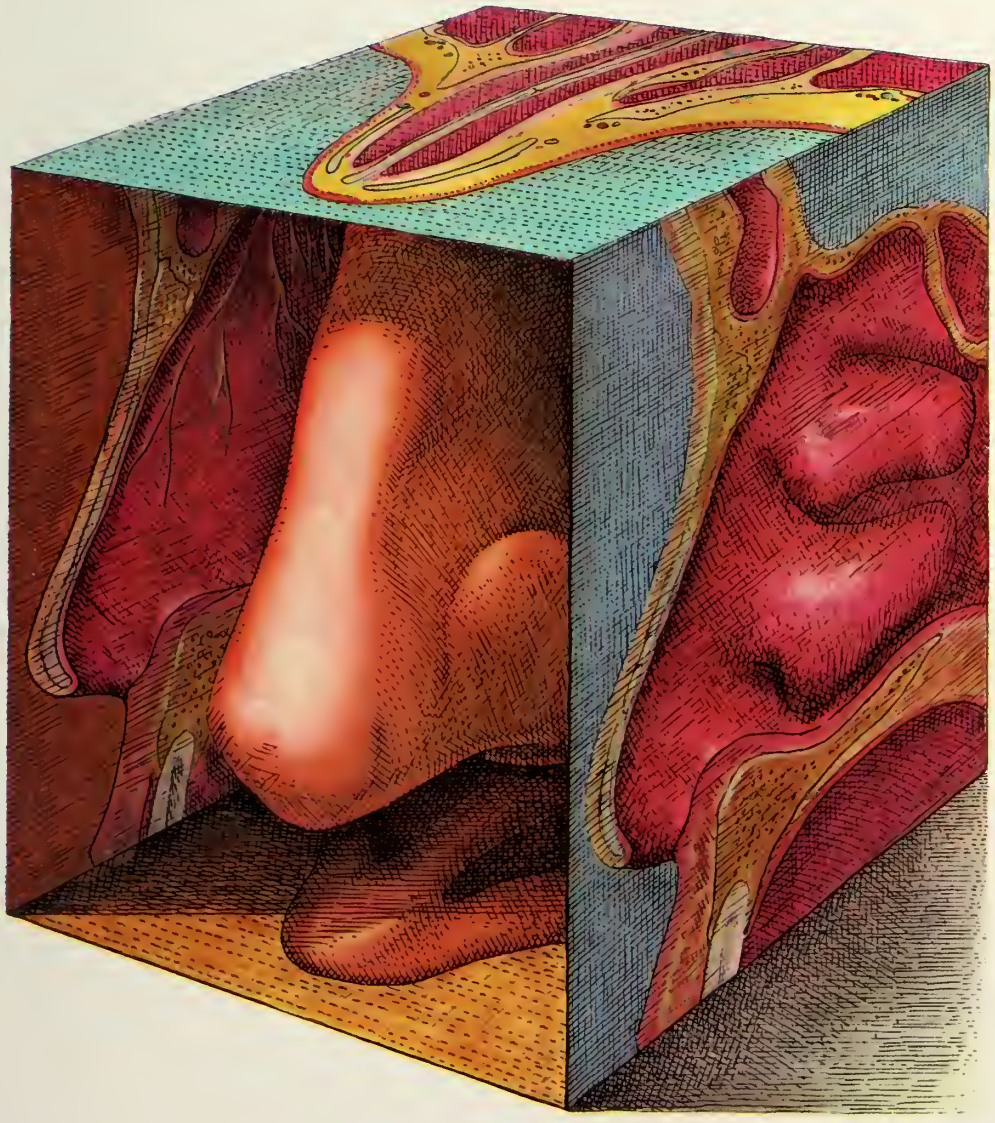
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spring 1967

DORSEY Season

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this issue: the ubiquitous world of summer allergies



the ubiquitous world of summer allergies

Donald L. Unger, M.D. • Clinical Assistant Professor, Department of Medicine (Allergy), Stritch School of Medicine (Loyola).

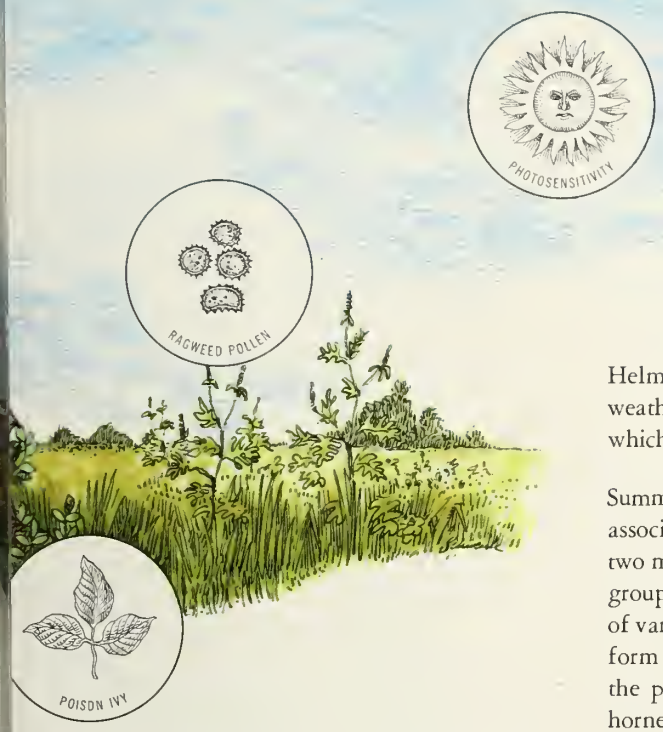
In the Spring a young man's fancy lightly turns to thoughts of—allergies. This is at least true of the 10% of the population who have hay fever and the 4% who have asthma.[1] The snow melts, the trees blossom and the noses run. Patients who were fine all winter may not be enthralled by the sight of the first robin or the blossoming of a crocus, for their appearances may precede the "sneezin' season."

Allergies in general can be divided into winter allergies and summer allergies. In the winter the main problems are inside the house: e.g. dogs, cats, dust and feathers. Houses in the northern half of the country become so dry that it becomes essential to add humidity to the home; this is a far cry from the damp summer months with the moldy basements and need for dehumidifiers.

Early in April trees begin to pollinate, with each tree having about a two week period of pollination. A particular patient may be sensitive to only one tree and thus have his hay fever for such a short time that he thinks he has a cold.[2] The entire tree season starts about April 1 and ends about Memorial Day, although all hay fever seasons are blurred and prolonged in the southern part of the country. Tree pollen is usually very heavy and a person may well have most of his exposure from those trees immediately surrounding his home.

Grasses pollinate from about May 15 until July 4, and cause "rose fever." Grass pollens are somewhat lighter and more buoyant than tree pollens, and are much more ubiquitous. While there are several varieties of grasses in the United States, they are so closely related antigenically that a person sensitive to one is generally sensitive to them all.[3] Thus, while the tree season is really several small seasons intertwined, the grass season will usually result in symptoms for a more prolonged period. Obviously, a grass-sensitive patient will have trouble only when grass is pollinating—he will have to think of another excuse not to mow the lawn after July 4.

Ragweed is the "Big Daddy" of them all in the eastern two-thirds of the country. Pollination is generally from mid-August until the end of September, with the predicted lower counts and longer seasons



in the southern part of the country. Ragweed is a very light pollen which may be windborne for hundreds of miles. An interesting study was made in New York City, in which 90% or more of the ragweed plants were destroyed in three of the five boroughs; pollen counts done during the season were virtually identical in all five.[4]

Ragweed is, of course, the most common cause of hay fever and is associated with an incredible loss of man hours from work each year. Many is the patient who travels to areas where the pollen count is low, just to avoid having symptoms. There is no ragweed anywhere in the world except the United States and portions of Canada and Mexico.

While molds are present through the year, the most important ones predominate from April until November. An old wives' tale has ragweed ending with the first frost, when actually it ends a good month earlier. It is Alternaria—the kingpin of the molds—that meets a sudden demise with the first frost. Alternaria-sensitive patients are in their glory when there is snow on the ground, and might be ideally suited to man the radar stations in Alaska. In September and October, Alternaria counts are at their highest, perhaps associated with the burning of leaves. Other molds such as Hormodendrum and Helminthosporium are associated with the warmer weather, as opposed to Penicillium and Aspergillus which are household molds.

Summer also means the return of our much maligned associates—bugs. Insects cause allergic symptoms by two methods: the bite or sting of the Hymenoptera group, and the inhalation of particles of the bodies of various insects. Wasp stings are the oldest known form of allergy, as they caused the death of one of the pharaohs in ancient Egypt.[5] Bees, wasps and hornets account for many deaths in this country, and those sensitive to them should carry special treatment kits at all times; a few minutes delay in the administration of epinephrine to such a patient, might be the difference between life and death. Inhalation of particles of insects may cause sneezing and wheezing in a susceptible individual.[6] Both of these forms of insect allergy may be benefitted by hyposensitization.

The insect recognizes no professional bounds. He is as apt to bite the physician as the patient. So this season, beware of bugs. And beware, too, of poison ivy. That pleasant stroll through the woods and underbrush with the Boy Scouts might turn into a

(Concluded on following page)

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nightmare for the botanically uninitiated in the causes of rhus dermatitis (poison ivy, poison oak and poison sumac). Although you may have been careful, your dog may not have noted that it wasn't clover he jumped through, but poison ivy. His return to your side may give you the rhus dermatitis that you so carefully avoided. That heavenly campfire may be emitting particles of rhus oil to produce an airborne contact dermatitis of the exposed areas of the body.

Another fascinating, but rather infrequent type of summer allergy is physical allergy. Some people sneeze on exposure to sunlight, while others break out in rashes, usually on the exposed parts of the body. These rashes may well follow the administration of various photosensitizing drugs, e.g. demethylchlortetracycline.[7] Another form of physical allergy and one that may be lethal in the summer, is cold allergy. Yes, I mean cold allergy, not heat allergy. The cool dip on a hot day with its consequent sudden chilling of the body, may be the coup de grace for a cold sensitive patient.[8] It is customary to write "heart attack" on the death certificate, even though the victim may have been an 18-year-old boy who looks like a Greek god.

Lest the reader be depressed by this saga of afflictions associated with the warmer months, perhaps he should remember that it is also a time for swimming, baseball, lying in the sun and taking that long-planned vacation. So let's all join in a chorus of "In the Good Old Summertime," as we sneeze, wheeze and scratch. Be careful of your suntan lotion, however; it may cause you a contact dermatitis.

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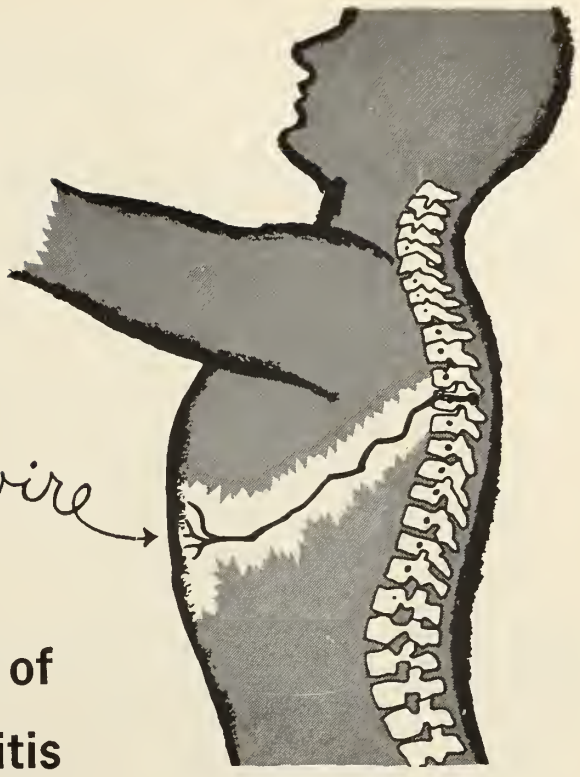
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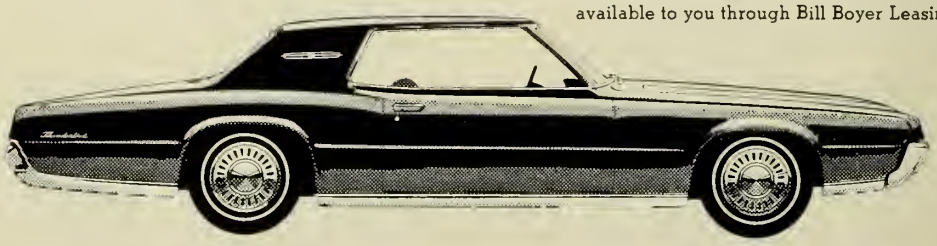
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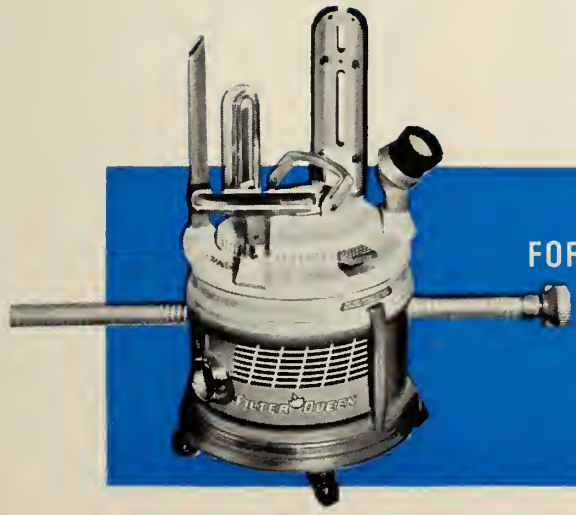
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The Journal-Lancet

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SYMPOSIUM — Physical Medicine and Rehabilitation — PART I

Physical Medicine and Rehabilitation as a Medical Specialty

FREDERIC J. KOTTKE, M.D.

Minneapolis

REHABILITATION has emerged as a key word in community programs for comprehensive medical care. The report of The President's Commission on Heart Disease, Cancer and Stroke and the subsequent federal legislation to enact these programs have emphasized the inclusion of rehabilitation medicine in all developing programs for medical care. The regional medical programs which are now in the planning stage will place an increased emphasis on physical medicine and rehabilitation. Medicare for the aged provides for physical medicine and rehabilitation in the hospital, extended-care facilities, and the home. In all of these ways rehabilitation medicine is extending into the practice of general physicians in every community. There will be an increasing demand on physicians to ascertain that comprehensive medical care is provided to patients either by direct participation of each physician or by referral to medical specialists when that is indicated. The series of articles in this issue of *The Journal-Lancet* presents a number of applications of physical medicine and rehabilitation in medical practice.

The origin of this field of medical interest dates back to the early 1920s, when the American Congress of Physical Therapy, X-ray and Radium was formed. Several years later the American Medical Association authorized the inclusion of the Congress in its list of special societies. Shortly thereafter, the Congress dropped the terms "X-ray and Radium" from its title, indicating that the educational program was now restricted primarily to physical therapy. In 1944 "physical therapy" became "physical medicine." This change was effected to distinguish between the interests of the physician and the role of the physical therapist. Diagnostic as well as therapeutic uses of physical agents and occupational therapeutic modalities were now included. By 1946 the Council of Physical Medicine of the American Medical Association sponsored the term "physiatrist" as the designation for the physician practicing physical medicine. The American Board of Physical Medicine was organized in November 1947 under the aegis of the Advisory Board for Medical Specialties. The name was changed to the American Board of Physical Medicine and Rehabilitation in 1949. The change came largely through efforts of Dr. Howard Rusk, who is often credited with adding the rehabilitation emphasis to what had been regarded a society of medical physicists.

The medical profession in Minnesota has been in the forefront of physical medicine and rehabilitation for many years. From the time of the pioneering specialization in physical medicine by Miland E. Knapp in Minneapolis and Frank H. Krusen at the Mayo Clinic, there has been a continually growing interest in physical

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medicine and rehabilitation as a medical specialty in Minnesota. The first three-year program of formal training in physical medicine and rehabilitation in the United States was offered at the Mayo Clinic in 1936. In 1945, the University of Minnesota instituted a three-year graduate school program in physical medicine and rehabilitation. Since then both Mayo Graduate School of Medicine and University of Minnesota Medical Graduate School have had approved residencies in physical medicine and rehabilitation.

The initial interest of physiatrists in the therapeutic efficacy of the modalities of heat, light, water, electricity, massage, and exercise was modified as physicians were confronted by large numbers of patients with chronic and disabling conditions and continued management became increasingly required. Accumulated experience in physical medicine and rehabilitation made it apparent that the application of therapy to a passive patient was not effective for restoring functional independence. Today, emphasis is upon the therapeutic use of exercise and other forms of activity to help the patient use his residual capacities to develop his optimal level of independent function. Prolonged illness with residual disability leads to diminution of psychosocial as well as physical capacities, and therapeutic activity is able to improve both. The disabled individual is helped to mobilize existing compensatory and adaptive mechanisms in both his internal and external environments in order to function more appropriately in his impaired state. This is called the rehabilitation process. Rehabilitation management of disability seeks to enhance this compensatory adaptation by every means: physiologic, pharmacologic, surgical, mechanical, psychologic, and sociologic.

The specialty of physical medicine and rehabilitation deals with the adaptation of the individual whose function has been impaired by disease, injury, or congenital malformation. Three decades ago the outlook for many with such disabling conditions was bleak. This field of special medical interest developed in response to the physician's frustration in dealing with the manifold problems of chronic disease and disability. A fresh approach was demanded which led to advanced concepts of medical care, reorganization of medical knowledge, and development of new knowledge and special technology.

Physiatry is concerned with the scientific study of the nature of physical disability as well as the evaluation and management of the patient whose impairment stems from dysfunction of mobility, communication, and social interaction. It includes goals of improved physical, psychological, social, and vocational functioning, with or without change in the underlying disease process. In order to maintain and improve function, the physiatrist must have the capacity to [1] evaluate disability and residual function (diagnosis), [2] set goals for ultimate function (prognosis), and [3] devise and supervise programs of restoration toward those goals (treatment).

The treatment of the many problems of the disabled requires the services of a variety of disciplines; stress is placed on the concept and practice of interdisciplinary team work. A well-integrated professional team is more likely to achieve the desired goals than are the combined, although independent, efforts of individual members. Physiatrists are trained to give medical direction and collaborative leadership to such a group, consisting of nurses, physical therapists, occupational therapists, speech therapists, clinical psychologists, rehabilitation counselors, and social workers. As well-trained clinicians, physiatrists are able to collaborate effectively with medical colleagues in achieving the goal of comprehensive medical care of the patient. Mobilization of family and community resources, so essential to the management process, is emphasized. Planning and implementation of continuity of restorative care is an appropriate medical responsibility; it bridges the gap between intensive treatment and social reintegration.

Because the physiatrist may have to draw upon a wide variety of health services and often makes use of elaborate and expensive equipment, he frequently practices either fully or part time in a medical center, rehabilitation center, or hospital. Almost 35% of all psysiatrists are employed as full-time staff members in voluntary and governmental hospitals. Approximately 40% of physiatrists are engaged in private practice. Opportunity also exists for the physician interested in an academic career. Nearly 20% of Board-certified physiatrists are full-time medical school faculty members; almost half of all physiatrists do some teaching in medical schools.

The demands for physicians trained in physical medicine and rehabilitation greatly exceed the supply. In all areas—clinical practice, rehabilitation centers, institutional programs, and medical schools—there are almost unlimited opportunities in every part of the United States. It is possible for a physician who decides to specialize in this field to select the type of practice he wishes to engage in and to find available opportunities. It appears that under federal programs these opportunities will be more extensive.

There are now approximately 800 physicians limiting their practice to physical medicine and

rehabilitation, of whom more than 500 are diplomates of the American Board of Physical Medicine and Rehabilitation. It has been estimated that there is a current need for at least 2,000 additional physiatrists. This does not take into account an anticipated expansion of demand to at least double this number. Two thousand additional physiatrists could be absorbed immediately by existing openings in hospitals, rehabilitation centers, medical schools, and communities with opportunities for private practice. The Veterans Administration alone reports openings for 160 physiatrists, including 17 chiefs and 11 assistant chiefs of physical medicine and rehabilitation services. In the 60 medical schools where the teaching programs in rehabilitation medicine are headed by physiatrists, openings exist for an additional 89 qualified physiatrists. From these few figures it can be seen that there is almost unlimited opportunity for specialists in this field. Despite growing recruitment, there is little prospect that the supply will approach the demand in the foreseeable future. There is also no doubt that future changes in patterns of medical care and in the age distribution of the population will accentuate the shortage of physiatrists. It is difficult to predict the impact of such developments as Medicare and legislation for heart disease, cancer, and stroke on the need for physiatrists. The question does not seem to be, "Will the need increase?" but, "By how much?"

Training to qualify for certification by the American Board of Physical Medicine and Rehabilitation includes graduate training in anatomy; physics, particularly of radiation, electronics, and instrumentation; physiology of the neuromuscular, cardiovascular, and pulmonary systems; pathology as it applies to disabling diseases; biochemistry with emphasis on the metabolism of disuse and activity; and such other basic sciences as are appropriate to the field. The examinations require knowledge of the physiologic effects and clinical uses of physical agents and of occupational therapeutic modalities; diagnostic procedures such as electrodiagnosis, manual muscle testing, goniometry, testing of activities of daily living and testing of peripheral blood flow; prosthetic, orthotic, and self-help devices; and techniques and roles of associated professional personnel and how to integrate them. Residency programs have bed services affording experience in continuing clinical management of adults and children who may be chronically ill as well as disabled.

During the past five years the American Board of Physical Medicine and Rehabilitation has accepted four years or more of general medicine practice in lieu of one of the three years of formal training required for certification by that Board. At the same time, the Vocational Rehabilitation Administration has offered advanced traineeships to doctors who have been in general practice for four years or more to return for formal training in physical medicine and rehabilitation. The experience with physicians returning from general practice to take specialist training in physiatry has been quite satisfactory. Doctors in general practice have had experience in the management of the personal and family problems of patients with chronic diseases. They have learned to understand the needs of these patients, and when they enter physical medicine and rehabilitation they are prepared to accept the broad responsibilities involved in the management of continuing disability.

For the interested physician, the specialty of physical medicine and rehabilitation offers an approach which is holistic rather than atomistic, comprehensive rather than fractional, and flexible rather than doctrinaire. It utilizes modern knowledge of the biology of adjustment to disability, its natural course, its pathologic complications, and its response to intervention. The properly motivated patient returning to his home and his community is not viewed as a happy accident of suitable premorbid personality characteristics but as a resultant of integrated therapies brought to bear simultaneously on all phases of the individual's treatment and his continuing life as a disabled individual.

In a field dedicated to the conservation of human resources, opportunity exists for those interested in patient-centered medical care, in academic and research careers, and in administration.

It is hoped that the opportunities for training in Minnesota, the availability of special traineeships for doctors returning from practice to specialize in physical medicine and rehabilitation, and the widespread opportunities for practice following training will encourage many more physicians to enter this medical specialty.

Both the Mayo Graduate School of Medicine and the University of Minnesota Medical Graduate School have approved fellowships in physical medicine and rehabilitation. Information regarding the opportunities for training and practice may be obtained from Mayo Graduate School of Medicine, Rochester, or Department of Physical Medicine and Rehabilitation, University of Minnesota Medical School, Minneapolis.

Practical Rehabilitation

GORDON M. MARTIN, M.D.

Rochester, Minn.

THE TERM "practical" is defined as that which is workable, feasible, or efficient. Thus, our subject, "Practical Rehabilitation," suggests a concept that at least some rehabilitation is workable, feasible, and economically sound; it also implies, however, that sometimes efforts at rehabilitation can be unworkable, unfeasible, and even doomed to failure. As for the term "rehabilitation," its meaning has become confused through overuse and misuse and some physicians have even grown to abhor the term. But relative to medical practice, a number of concepts and definitions of rehabilitation have evolved, all of them perhaps endowed with various degrees of common sense, idealism, and usefulness. Some of these definitions are as follows: (1) Rehabilitation involves treatment and training of the patient to the end that he may attain his maximal potential for normal living—physically, mentally, socially, and vocationally.[1] (2) Rehabilitation is the important third phase of medicine, the period of management after the acute period—between the bed and the job. (3) A more philosophic definition states that rehabilitation is a bridge spanning the gap between uselessness and usefulness, between hopelessness and hopefulness, between despair and happiness. (4) In a more mundane vein a physician has said that everything he does for all of his patients to get them well and back on the job is rehabilitation.

In setting the stage for a discussion of practical rehabilitation, let us look, for an analogy, at another relatively new and intriguing field, that of cardiac surgery. If a surgeon were to discuss the topic, "Practical Cardiac Surgery," the following basic principles and conclusions would become obvious. (1) Many heart operations that were impractical ten years ago are now practical, (2) cardiac surgery that may be performed successfully by a trained and experienced cardiac surgeon is practical, while similar surgery would be impractical for most other surgeons, (3) heart operations that are feasible in a hospital with special equipment and facilities for heart-lung bypass, hypothermia, elaborate monitoring devices, and other special techniques would be impractical or impossible in many general hospitals, (4) accurate recognition of a cardiac lesion or abnormality that structurally lends itself to repair or reconstruction is a determining factor in deciding whether an operation is practical, and (5) an operation that might be practical if performed on a child or youth might be completely impractical if done on an older patient.

Similarly, our decision regarding the practicality of rehabilitation for a given patient rests on several significant factors. Although successful rehabilitation of some patients can be accomplished in a community general hospital, frequently it is advisable to transfer a patient to some other hospital or to a rehabilitation center. The institution selected should have special areas and organization, including the necessary equipment and physicians, nurses, and paramedical personnel with special training and experience in the essential rehabilitation procedures. Unfortunately, a physical therapist working alone or with an aide in a small physical therapy department in an average community hospital cannot be expected to provide adequate rehabilitation services for the seriously handicapped and disabled patients. Whether or not he will provide or supervise rehabilitation, it is the responsibility and prerogative of the patient's general physician to decide the practicality of rehabilitating a patient with chronic disease and disabilities.

Some ten years ago rehabilitation programs were concentrated on patients with residual effects of poliomyelitis, patients with paraplegia, some with cerebral palsy, and a few with hemiplegia. Fortunately, the number of patients with residuals of paralytic poliomyelitis has been strikingly decreased. At present the rehabilitation of paraplegic and hemiplegic patients is considered almost routine in more and more places. In recent years we have been seeing a

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growing interest in the rehabilitation of patients with other disabilities. Some of the areas that are now being explored and developed as fields for practical rehabilitation include traumatie quadriplegia, multiple sclerosis and other demyelinating diseases of the central nervous system, parkinsonism, pulmonary emphysema, degenerative and rheumatoid arthritis, posttraumatic brain syndromes, metastatic malignant disease, cardiac disorders, and amputations. Although this is a partial listing, it will be possible to sample only a few areas, some newer as far as the applieation of rehabilitation concepts are concerned, and some older but still neglected.

Some of the conditions which we are now encouraged to tackle more optimistically were formerly considered hopeless and progressive in their course; and, heretofore, for some patients who were considered too old or who were not wage earners, it was thought economically unsound to spend relatively long periods of time at considerable expense in efforts to achieve perhaps only very modest goals. However, with the development of more centers, more trained personnel, more understanding and know-how on the part of physicians, and better eeonomie resources through group or private insurance, Workmen's Compensation, and vocational rehabilitation funds, rehabilitation as a specific part of the treatment program becomes feasible.

THE STROKE PATIENT

The importance of praetical rehabilitation for the stroke patient is rapidly beeoming apparent. At the recent National Stroke Congress many facets of the stroke problem were analyzed and discussed. The increasing incidence of stroke syndromes and the importance of exploring various aspects of prevention were eonsidered. However, the greatest emphasis of the Stroke Congress was on the development and use of effective programs for treatment and rehabilitation.

Until recently, many physicians held a nihilistie view toward prolonged treatment and rehabilitation of the hemiplegic patient. Too often the patient was dismissed from the hospital two weeks after an episode of cerebral thrombosis with hemiplegia. His further recovery was left to a somewhat pessimistic wateh-and-wait attitude on the part of the patient, his family, and the physician. The patient all too frequently remained a nursing-care problem with supportive treatment based only on a few suggestions from a visiting nurse or from the public health booklet, "Strike Back at Stroke."

The physical treatment and rehabilitation of the hemiplegic patient should begin within the first week after the stroke and be continued on a regular, supervised, intensive basis as long as progress is being made. It has been estimated that approximately 80% of stroke patients ean be relatively well rehabilitated, independent, and usually walking in four to eight weeks after a stroke. It is unfortunate that many patients are dismissed to their homes—perhaps in order to free a needed hospital bed—just at the time when intensive efforts should be made in their rehabilitation. Often before the end of the first week after a stroke the patient can be in a wheelchair, learning to feed himself and to perform much of his own bodily care with the use of his normally functioning arm and hand. The morale of these patients remains higher if early and active efforts are made in getting them out of bed and starting a positive treatment program.

Although a small hospital physical therapy department may not have all of the facilities of the larger rehabilitation center, most physical therapists, with the aid of an understanding and guiding physician, can do a ereditable job in the physical rehabilitation of the stroke patient. A problem that often presents itself, however, is that the lone physical therapist is often burdened with too many patients and is not able to devote adequate attention to each one. As a result, the older hemiplegic is perhaps unfairly eonsidered to be a ehronic case and not a good candidate for rehabilitation.

A brief review of the steps of the rehabilitation of the average hemiplegic patient would include, in early eare, the proper positioning of the patient and the use of a simple spring-wire foot-drop splint to prevent development of a tight heel cord. Early passive exercise of the involved extremities ean usually be started three or four days after the stroke. As function returns, reeducation and active assistive exercises are undertaken. Generally during the early treatment period, particularly if the patient has had a completed stroke, the patient can achieve standing balance by means of temporary splinting for the paralyzed leg. Assisted walking in parallel bars is undertaken as soon as he is able to flex the leg or hike the hip and has an adequate sense of standing balanee. As soon as possible, standing and walking practice should be undertaken many times a day and not just at a single treatment session.

Nurses and oceupational therapists should encourage and help the patient to learn to feed himself early, to care for his bodily needs, to dress and bathe himself, and to move from bed to wheelehair and from wheelchair to toilet with minimal assistance. Although a catheter may be

needed for the first few days after a stroke, generally, as the patient becomes cooperative and aware of this problem, he can soon relearn and redevelop normal bladder and bowel function. The majority of hemiplegics who recover partial function of the lower extremity, although they may have little or no return of upper extremity function, can learn to walk with a glider cane or regular cane plus a short leg brace.

Speech problems, whether they involve varying degrees of aphasia or merely problems of dysarthria, should be evaluated by a speech pathologist; and efforts should be made by a speech therapist to help these people achieve maximal effective speech, since good communication is one of the most essential parts of a successful rehabilitation program for a stroke patient. However, even severely aphasic patients deserve a trial period with a speech therapist during early convalescence, although little or nothing may be achieved with a few patients who have global aphasia.

We recently dismissed a 69-year-old hemiplegic patient from an intensive program of physical rehabilitation on a hospital physical medicine and rehabilitation service. Immediately after his stroke he was hospitalized in a very small community hospital which had no personnel or facilities for rehabilitation. Two weeks after the stroke he was dismissed to his home and remained a bed patient and nursing-care problem with essentially a complete left hemiplegia. After he had been at home two weeks, the family realized that he was regressing rather than progressing. He was then hospitalized in a larger community hospital where physical therapy was available. His physical therapy consisted of passive exercises to the paralyzed extremities once daily. During a two-week period he had been helped to stand in parallel bars on only two occasions. Six weeks after the stroke he was transferred to a hospital with equipment and facilities for an intensive rehabilitation program. At this time he was still bedridden. He had sat up only a few times. He had an indwelling catheter and partial incontinence of the bowels. He was started on a program of reeducation exercises twice daily; standing balance was learned on a tilt table; within a week the catheter had been removed and he had full control of his bladder and bowels. Hip flexor and quadriceps power was returning to the lower extremity, and soon gait training was undertaken in the parallel bars with the use of a gutter splint, foot-drop support, and sling for the paralytic arm. At the end of six weeks, or three months after the stroke, this patient was able to walk with a regular cane and short leg brace, care for his own bodily functions, dress himself, and be essentially independent around his home. The patient had already been retired, and he was content to continue on a retired status after the stroke. It is highly probable that an intensive program of rehabilitation begun immediately after the stroke would have achieved the same degree of function and independence with a shorter period of hospitalization and less cost to the family and the insurance company.

PARKINSON'S SYNDROME

New interest in the treatment and rehabilitation of patients with Parkinson's syndrome developed after the success of the surgical thalamotomy procedures first reported by Cooper about ten years ago. Although these procedures have proved extremely helpful to many patients with parkinsonism, it is estimated that they are applicable to only 10 to 20% of all the patients with this syndrome. However, often with a program of medication and regular therapeutic exercises much can be accomplished in prolonging the months or even years of activity and productivity. It is most effective when the patient is instructed in an exercise program in the earlier stages of his disease. The exercises are basically simple. They should be performed 2 or 3 times daily for short periods at home, and their aim is to maintain or increase mobility and speed of movement in muscles that are becoming slower and more rigid. The tendency to flexion-rigidity postures is combatted by emphasizing extensor movements of the peripheral joints and extension of the hip and trunk. Frequent short walks with careful attention to proper posture, arm swing, and avoidance of the shuffling, festinating gait are emphasized. The patient should be rechecked regularly at intervals of several months, because medication may need changing and exercises should be reviewed. In the late stages of parkinsonism most efforts at rehabilitation are not successful, although some limited degree of independence and increased mobility can be established with a period of active physical treatment and changes or adjustments in medication plus, in selected cases, thalamotomy. The rehabilitation achieved after early recognition of the syndrome is being recognized as a practical approach to a disease that was once considered progressive and not amenable to treatment.

DEMYELINIZING DISEASES

Patients with multiple sclerosis and other demyelinizing diseases of the central nervous system that are characterized by varying pictures of

weakness, spasticity, and incoordination can often achieve real benefit from specific efforts at physical rehabilitation. Again, early diagnosis and recognition of the problems together with establishment of an active and aggressive treatment program are more rewarding than watchful waiting and the application of late measures. Varying pictures of weakness, spasticity, and incoordination are seen. The patterns and degree of paresis are variable. Difficulty in walking is often due to marked weakness of the abdominal muscles and hip flexors. Foot drop may occur early and spasticity and clonus of the calf muscles may interfere with walking. A specific program of therapeutic exercise should be prescribed early and should be designed to maintain and improve strength of the paretic muscle groups. When ataxia is a problem, coordination exercises are employed and the patient learns to use visual reflexes and cues to help the coordination of movements of the lower extremities both in doing exercises and in walking. Often the use of a short leg brace, a cane, or German crutches may improve stability in walking so that there is much less danger of falling. This may permit the patient to carry out more activities at home, in society, and even on the job. Since the neurologic picture may be a changing one, these patients should be evaluated from time to time and appropriate adjustments should be made in their treatment program. Recently, some encouraging reports have been made of apparent improvement after intrathecal administration of corticosteroids. Obviously, some patients with advanced degenerative processes are not suitable candidates for rehabilitation, and practical results would not be obtained. The decision of whether something can be achieved should rest on decision of more than one physician.

PULMONARY EMPHYSEMA

The increasing frequency of severe and disabling pulmonary emphysema has been recognized by the medical profession during the last few years. At first a nihilistic attitude was taken by most physicians regarding efforts to rehabilitate these patients by means of an intensive multifaceted treatment program. Although this disease process is progressive, it appears that many patients, by following a rigid regimen, can increase their general physical activity and perhaps prolong their working years; in addition, it is possible to reduce the incidence of early fatal acute pulmonary infections. The basic program is established after evaluation of the patient's respiratory function and the severity of the associated chronic bronchitis. Such a program consists of (1) having the patient stop smoking, (2) having him use bronchial dilators, such as isoproterenol (Isuprel®), (3) training the patient in new breathing patterns which emphasize the expiratory phase of respiration through careful training in the use of the abdominal muscles with relaxation of the diaphragm combined with deep inspiratory breathing; in addition, proper postural habits are emphasized and combined with efforts to increase the walking distance gradually while the new breathing patterns are employed, (4) providing for postural drainage when indicated, (5) prescribing potassium iodide or other expectorants, (6) having the patient avoid exposure to respiratory infections and irritants, (7) providing early antibiotic treatment of respiratory infections, and (8) using positive-pressure oxygen for some patients as needed. The full and enthusiastic cooperation of the patient is required if any practical results are to be obtained. This is best achieved when the program is presented by the physician in a direct, positive, honest, and reassuring manner.

DEGENERATIVE JOINT DISEASE

Practical rehabilitation can frequently be achieved for many patients with crippling and disabling degenerative joint disease. This requires the early establishment of realistic goals. The most disabling degenerative joint disease usually occurs in the hips and knees. When the knees are involved, few orthopedic surgical procedures are available to improve or restore their function. However, for patients with involvement in one or both hips, a number of variably successful arthroplasty procedures have been devised and are in fairly common use. For those who are candidates for these procedures, the postoperative physical rehabilitation is most essential for strengthening the muscles, maintaining or improving joint range, and achieving practical ambulation techniques. These generally require several months of regular daily exercises at home by the patient himself and frequent reevaluation by the physicians and surgeons involved in his care. When surgical reconstruction is not indicated or feasible, the principal aim of the rehabilitation is to teach the patient the best ways of protecting the progressively degenerating joints. This may include the earlier use of a cane or crutches than the patient would be likely to consider on his own. Thorough instruction of the patient in restricting his weight-bearing activities is essential and may require a change to a more sedentary occupation. Often a direct attack on obesity is of prime importance. Too often these patients attempt to force their degener-

ating joints to maximal physical effort in walking and at work and want to rely on an occasional "shot" to help keep them going.

RHEUMATOID ARTHRITIS

Practical rehabilitation for the patient with rheumatoid arthritis must, of course, emphasize early adequate treatment in an effort to preserve function, to preserve the integrity of joints involved in a rheumatoid process, to maintain adequate muscle strength, and to minimize the development of disabling deformities. The reliance on suppressing rheumatoid inflammation by steroids plus the forced overuse of joints with active synovitis is not advisable. Adequate rest and protection of the involved joints, simple exercises to maintain motion and strength, salicylates for the reduction of pain, and, when necessary, suppression of the inflammatory reaction by administration of gold or small doses of steroids is the basic conservative program in the active phases of the disease. Recently, orthopedic surgeons have shown a renewed interest in rheumatoid arthritis, and effective reconstructive procedures have been devised for correcting deformities of the hand and have improved both function and cosmesis. Synovectomies, particularly in the knee joints, are apparently proving satisfactory for some patients.

For patients with developing deformities and various degrees of damage of joints, a period of three to twelve weeks on a hospital regimen will result in control of and considerable improvement in the patient's status. This may include regular physical treatment, the use of splints to help correct deformities, and carefully controlled medication and activities. Since most patients with rheumatoid arthritis have been active housewives, the vocational rehabilitation problem is aimed primarily at returning these patients to their families so that they can care for themselves and also resume as many of their household duties as is possible or as is compatible with the stage of their rheumatoid arthritis.

METASTATIC MALIGNANT DISEASE

Practical rehabilitation for patients with metastatic malignant disease is more and more becoming a recognized part of the treatment of these patients. We now know that much can be done for the patient with postmastectomy lymphedema even in the stage in which metastatic lesions are present. The volume of the heavy, painful, edematous, useless upper extremity can be reduced by physical measures, and the reduction can be maintained at home by wrapping the limb with elastic bandage or elastic sleeve and by maintaining regular periods of elevation. The patient with metastatic carcinoma in the pelvis or femur can usually continue ambulation safely if he is properly fitted and trained in the efficient use of crutches. Patients who have had pathologic fractures of the pelvis or femur also can often be rehabilitated so that they can be ambulatory. Patients who have had brain tumors or metastatic lesions in the brain can often be rehabilitated much as hemiplegics would be. The general debility and weakness which often develop in these patients can frequently be corrected by improving the general nutrition and by prescribing a guided program of mild strengthening exercises, ambulation, and wheelchair activities which will keep the patient active. With the availability of newer forms of antimetabolic drugs, more extensive surgical procedures, and more powerful types of radiation therapy, many of these patients can look forward to even relatively prolonged periods of improvement. Even patients with osteogenic sarcomas who have had amputations can generally be fitted with a prosthesis and may have a number of years of useful, nearly normal activity.

TRAUMATIC QUADRIPLEGIA

Traumatic quadriplegia is a major catastrophe. Treatment and rehabilitation are difficult at best. Complications are common and periods of depression and discouragement are unavoidable. The greatest asset is an inherent good to superior intelligence. Even with this advantage the patient needs encouragement and guidance from the physician and all others concerned with his treatment and rehabilitation.

A review of the treatment and rehabilitation of a youthful quadriplegic patient will illustrate the effort and time required for success.

CASE REPORT

A 17-year-old boy was injured in a pickup truck accident while on a summer job in July 1958. He was quadriplegic from the level of C-5 downward. X-ray films showed multiple fracture dislocations of the cervical portion of the spinal column. Two days after the accident some function returned to the elbow and wrist but none to the fingers. A cervical laminectomy was performed ten days after the accident; an extradural hematoma was removed and the spinal cord was decompressed. Following this, recovery of finger extension occurred. Early physical therapy was started. In September he was transferred from the hospital to a rehabilitation center, where function of the hand improved further and very slight function returned to the trunk. Within two months he was able to stand and take a few steps with braces and crutches. Some assistive devices for holding utensils and a toothbrush were constructed. In November 1958 he was admitted to a hospital with a C-7 quadriplegia and spastic paraplegia plus marked weakness and atrophy of most of the hand and forearm muscles supplied by

C-8 and T-1. The sensory loss to pain, temperature, touch, position, and vibration extended to the C-8 dermatome. The patient had a neurogenic bladder and required a urethral catheter.

The patient underwent an intensive program of strengthening exercises twice daily for the muscles of the shoulder girdle, upper arm, wrist, and finger extensors. In February 1959 multiple tendon transfers were performed on the right hand as follows: (1) extensor carpi radialis brevis to abductor pollicis longus, (2) extensor carpi radialis longus through a loop or tunnel for thumb opposition, (3) pronator teres to flexor digitorum profundus, and (4) brachioradialis to flexor pollicis longus. The purpose of the procedures was to maintain strong wrist flexion and extension, and to add the function of pinch and grasp; expendable muscles were used to provide this motor action. He left the hospital in May 1959 for graduation from high school and then was readmitted for multiple tendon transfers on the left hand, similar to those done on the right. These were functioning well at the time of dismissal from the hospital in August 1959.

In June 1960 he was readmitted to the hospital because of hyperextension deformities of the metacarpophalangeal joints of both hands. Further tendon surgery was performed on the right hand, including transfer of the extensor digitorum communis into the extensor pollicis longus to give better thumb extension, and a Riordan tenodesis was performed on both hands to limit extension at the metacarpophalangeal joints.

A Shipley-Hartford psychometric test indicated an IQ of 126. The Minnesota Multiphasic Personality Inventory indicated that the patient had some degree of unrealistic reaction to his disability. The patient was thought to be capable of college level work. His father suggested setting him up in business with a record shop. However, the patient chose to go to college and he graduated with above-average grades in June 1964.

In July 1964 he returned to the hospital for further surgery on the hand in an attempt to improve the function. An arthrodesis of the interphalangeal joint of the right thumb and transfer of the extensor pollicis brevis into the extensor carpi radialis brevis plus release of the proximal flexor pulleys and advancement of the volar plates of the index, long, ring, and little fingers were performed to correct the tendency to clawing, improve stability of the thumb, and improve pinch and grasp movements. Tendon transplants were functioning well at the time of dismissal. The patient has now entered law school.

Although the patient has not continued to do any practical walking, he is well adjusted to his disability and is completely independent physically as the result of wheelchair experience and the tendon-transfer procedures. He requires an indwelling catheter despite efforts to develop an automatic bladder. He takes sulfisoxazole (Gantrisin®) daily for one week out of every month to prevent infection of the urinary tract.

Maximal rehabilitation of a quadriplegic is obviously expensive and prolonged. Following his early hospitalization and rehabilitation, he has had 4 admissions to a hospital for a total of two hundred eighty days. The cost of hospitalization was approximately $8,000. The cost of medical, surgical, and physical medicine and rehabilitation services was about $6,000. Previous surgery and hospitalization during the period immediately after the accident can be roughly estimated at another $4,000, for a total of about $18,000. The costs were met in large part by Workmen's Compensation coverage, by insurance, and in part by the patient's family.

Is such complex and costly rehabilitation practical? If this young man becomes a successful lawyer, and an independent, self-supporting citizen for the next twenty to forty years, certainly this investment of money, time, and effort is small compared to the cost for care and maintenance of a neglected, unrehabilitated quadriplegic.

ECONOMIC FACTORS IN PRACTICAL REHABILITATION

Practical rehabilitation is now becoming a reality, or at least a possibility, for an increasing number of patients seen in general medical practice. The economic advantages of rehabilitation efforts have been proved again and again. Several years ago financial factors precluded rehabilitation in many patients. However, at present, funds are available for many who are unable to finance their own rehabilitation. Basic health insurance plans written by private companies for a family provide coverage usually for ninety to one hundred twenty days. This assures adequate periods of hospitalization for a large percentage of patients suffering from the conditions I have described. Other policies that are becoming more popular include major medical coverages up to $10,000 and comprehensive major medical plans, at somewhat lower premiums, which will give coverage up to $10,000 to $15,000 over a period of three years.[2] These insurance programs are adequate for most of the severely handicapped and disabled patients such as those with paraplegia, quadriplegia, and advanced arthritis. The Workmen's Compensation Laws,[3] which are now in force in all states, are realistic insurance plans to assure injured and disabled workmen adequate medical care and rehabilitation in addition to a salary allowance. The Division of Vocational Rehabilitation of the Department of Health, Education, and Welfare, working through state and area offices, has funds to provide for the medical and surgical aspects of rehabilitation and has personnel to evaluate and guide handicapped patients through educational and sheltered workshop programs and into suitable employment commensurate with their disability and handicap. The Kerr-Mills Law, now active legislation in most states, provides funds to aid in the rehabilitation of the patient who is more than 65 years old and whose own resources plus those provided by Medicare may be too limited to permit the necessary medical and rehabilitation care.

SUMMARY

Our concepts of rehabilitation of patients with neuromuscular and skeletal disabilities have undergone a significant evolution during the past decade. Now many patients with such dis-

(Continued on page 122)

Osteoporosis

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Osteoporosis may be thought of as a disease of insufficient bone and is to be differentiated from osteomalacia. Osteoporosis implies bone of normal composition but with insufficient volume. Osteomalacia implies bone of sufficient volume but with insufficient mineral phase. Osteoporosis affects an estimated 4 million people in the country and is four times more common in females than in males.[1]

The word osteoporosis is a nonspecific, descriptive term describing the anatomic and roentgenologic appearance of the diseased bone. As such, it may be seen as a primary or idiopathic disease as well as a secondary process resulting from some underlying abnormality.

CAUSES OF OSTEOPOROSIS

The most common form of the disease is the so-called senile or postmenopausal variety, which is, to date, without any clearly defined etiology.

Osteoporosis may also be seen in association with an established endocrinopathy. The most familiar example of endocrine-related osteoporosis is seen in association with hypercortisonism of either endogenous or exogenous origin. An unusual feature of osteoporosis associated with hypercortisonism is that on the roentgenogram it may occasionally appear to heal when the excess hormone is removed, especially in children. However, healing usually leaves behind a core of collapsed, osteoporotic bone. Another well-recognized form of endocrine-associated osteoporosis is the bony atrophy associated with hyperthyroidism. In this disease, osteoporosis results from excess breakdown of bone compared to formation of bone. Hyperparathyroidism is, of course, a well-recognized cause of osteoporosis, in addition to being the cause of von Recklinghausen's disease, or osteitis fibrosa cystica. An interesting feature of this form of osteoporosis is that extirpation of the hyperplastic parathyroid gland causes the cystic lesions to regress but does not influence the osteoporosis.

Nutritional deficiencies may also lead to osteoporosis. Scurvy, or vitamin C deficiency, may produce a wide spectrum of disease, ranging from simple bony atrophy to severe osteoporosis and osteomalacia. This phenomenon was seen in a large group of individuals with "hunger osteopathy" from the prisoner of war camps during World War II. Dietary calcium deficiency has also been recognized as a cause of osteoporosis.

Some of the other more common forms of secondary osteoporosis include immobilization osteoporosis, subchondral osteoporosis in association with rheumatoid arthritis, osteoporosis associated with neoplasm, and bony atrophy associated with some anemias. Gaucher's disease, acromegaly, multiple myeloma, osteomyelitis, and osteogenesis imperfecta are other causes of osteoporosis, although they are less frequently seen.

DIAGNOSIS OF OSTEOPOROSIS

The X-ray picture of osteoporosis is important to recognize, for it is by X-ray alone that the clinical diagnosis of osteoporosis is made in the majority of cases. Commonly, the disease affects the spine and pelvis, leading to a narrowing of the end-plates of the vertebral bodies. The vertebral bodies become biconcave, and the disk spaces take on the typical "fish mouth" appearance of the osteoporotic spinal column. Vertical stress-bearing trabeculae become prominent in comparison with horizontal-stress bearing trabeculae. The cortices of the vertebral bodies become progressively thinner and may eventually collapse. A curious observation is that spinal osteoporosis is usually associated with an absence of degenerative spurring of the spine, so commonly seen in elderly patients. Conversely, it is usually associated with the presence of abdominal aortic calcification. Individual bones may vary in a single patient, or different areas of the same bone may manifest radiologic presence or absence of osteoporosis.[2]

Over the last twenty-five to thirty years, much

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has been done in the field of roentgenology to make the diagnosis of osteoporosis more specific.[3] Such tools as graduated wedges of ivory or aluminum, cadaver bones of known mineral content, and graduated solutions of calcium chloride have been tried. These standards can be exposed to the roentgen ray on the same film beside the bone in question, and an attempt can be made to quantitate the amount of osteoporosis. However, these "specific" techniques have generally fallen under the heel of scepticism, which stems from recognition of the multiplicity of variables involved in such a process.[4] It would seem that bone densitometry has yet to elevate itself from the experimental field to clinical practice.

PATHOGENESIS

In order to approach the problem of the pathogenesis of osteoporosis, it is necessary to understand the metabolism of normal bone. Among its other unique characteristics, bone is the body's storehouse of calcium. It contains over 98% of the body's calcium stores and, as such, it is called upon from time to time to supply calcium from these stores. Calcium itself is essential to the body in many respects. It is necessary for normal neuromuscular excitability, blood clotting, cardiac function, and muscular contraction. The body gets its supply of calcium from dietary intake. In the event of inadequate dietary calcium, the deficit must be made up either by diminished excretion or by resorption from bone. Also, when persistent or abnormal amounts of calcium are lost in feces, urine, or milk and dietary intake is not sufficient for replacement, bone resorption must ensue to maintain a normal internal milieu.

Bone formation. Osteoporosis may be thought of as a disease of decreased bone formation. The role of the gonadal hormones in bone formation has been strikingly illustrated in birds, especially the pigeon. Estrogen given to pigeons results in an increase in the serum calcium concentration and a concomitant increase in bone formation. However, this hormonal effect is not the result of estrogen alone, since the castrated male pigeon does not show this phenomenon unless he is supplied with exogenous androgen.[5] In mammals, however, the effect of sex hormones on bone formation is less striking. No increase in serum calcium occurs with prolonged administration of gonadal hormones. Whereas it is true that administration of estrogens to young mice results in a decrease in bone resorption and an increase in bone formation, prolonged use of androgen and estrogen leads to a secondary retardation in bone formation with resultant osteoporosis. Older mice similarly treated, on the other hand, show no increase in bone formation and, in fact, show an increase in resorption and a decrease in bone formation with prolonged administration of sex hormone.

Based upon observations in animals, Albright and co-workers[6,7] treated a group of osteoporotic patients with estrogens and showed not only symptomatic relief but also calcium retention as well. Since then, others have confirmed the effect of estrogens on osteoporosis in human beings. However, one of the principal criteria for effectiveness of any form of treatment of osteoporosis is symptomatic relief. Solomon and associates[8] showed rather convincingly that the relief of pain and the subjective sense of well-being with estrogens is a placebo phenomenon. Yet it should be remembered that after menopause 17-ketosteroid excretion falls while corticosteroid production remains essentially unchanged. This suggests that the anabolic-catabolic balance of normal adulthood may become upset with increasing age.

Bone resorption. A second possible mechanism for the pathogenesis of osteoporosis is increased bone resorption. In the event of insufficient dietary calcium, inadequate intestinal absorption of ingested calcium, or persistent abnormal excretory losses, bone must make calcium available to the body's chemistry. If an inordinate amount of calcium is either denied to or lost from the body through any one of these routes, bone resorption must ensue, unless the body can adapt either by increased absorption or by decreased excretion of calcium. This type of adaptation is not uncommon in normal physiology. The lactating female adapts to the calcium loss in breast milk by increasing intestinal absorption. Perhaps the 25 to 30% of the population with osteoporosis consists of individuals who cannot adjust to decreased dietary intake, are able to absorb only limited amounts of calcium, or persist in abnormal excretory losses.[9]

Dietary calcium. Thorangkul and associates[10] compared phalangeal radiodensity of subjects on habitual dietary calcium intakes greater than 1,250 mg. per day with subjects on habitual intakes of less than 750 mg. per day and found a greater density in the subjects with the higher intakes. Schraer and Neuman[11] showed a similar difference in the second phalanx of the fifth digit in a group of adolescent South American Indian boys on low calcium diets who were compared with a peer group of adolescent American boys on higher calcium intakes. Vinther-Paulsen[9] measured the habitual dietary calcium intake of a group of elderly people and found that as many

as 74% of those ingesting less than 0.5 gm. of calcium per day had osteoporosis. The incidence of osteoporosis in elderly people with dietary intakes greater than 0.5 gm. of calcium per day was only 14%. Several other workers have shown a basic difference in diets of normal individuals and patients with osteoporosis. Elderly people with osteoporosis eat less calcium than do normal individuals.

The question of adaptation to a deficiency in calcium intake or an excess in calcium loss has been studied by Malm.[12] A group of males of varying ages who took 940 or 450 mg. of calcium per day was studied for a prolonged period. On the high calcium diet all were in positive balance by an average of 78 mg. per day. The subjects on the low calcium diet varied in their balance with a mean negative balance of 33 mg. per day. Some subjects developed a persisting negative calcium balance on the lower diet, while others with negative calcium balances eventually adapted, bringing the negative calcium balances to normal.

Can one achieve a more positive calcium balance by increasing the dietary calcium? Most attempts to answer this question have been based upon dietary survey techniques and analyses of representative diets and excretions. Bogdonoff and co-workers[13] conducted complete calcium balance studies on a group of elderly men and showed that the normal men achieved calcium balance at a dietary intake of 0.6 to 0.8 gm. of calcium per day. The osteoporotic patients achieved a positive balance only with an increased intake of 1.5 gm. of calcium per day.

Harrison and associates[14] studied 16 postmenopausal and senile osteoporotic individuals and compared them with 12 normal subjects. Six of his patients with osteoporosis were studied for nine to forty-eight months on a high calcium diet. In that study, patients with osteoporosis developed a more positive calcium balance than did normal subjects at a similarly high intake, and 4 of the 6 studied for a prolonged period remained in strongly positive calcium balance.

Nordin[15] studied 17 patients with osteoporosis on 2 levels of dietary calcium for twelve days on each level. Their usual dietary intake was 17.7 mg. of calcium per kilogram of body weight per day. At this level they were in a positive calcium balance of 2 mg. per kilogram per day. After one week of taking 35.3 mg. of calcium per kilogram of body weight per day, calcium balance was increased to 9.4 mg. per kilogram per day. Several patients were studied for more than a year at a dietary intake of 44.7 mg. of calcium per kilogram of body weight per day. These subjects remained in a positive calcium balance of almost 16 mg. per kilogram per day.

Lutwak and Whedon[1] continuously measured calcium balance with various calcium diets in patients with postmenopausal and senile osteoporosis. They concluded that all of their subjects showed an increased calcium balance as dietary calcium increased. After a period of time, however, some patients showed adaptation with a progressive decline in the amount of calcium retained. The intake at which calcium balance occurred varied widely but was greater than 0.8 gm. of calcium per day in all subjects. Some required an intake of greater than 2 gm. per day to produce a positive calcium balance. These authors also studied the effects of estrogens and estrogens together with androgens in patients on varying calcium diets and found no increase in calcium balance using these hormones.

A somewhat different approach to the study of available calcium was made by Haas and others.[16] They studied calcium retention in normal and osteoporotic patients without hypercalcemia by means of intravenous calcium infusions. They then related the serum calcium concentration to serum alkaline-phosphatase activity and concluded that a group of 34 osteoporotic patients had an increased avidity for calcium relative to their decreased bone mass. They suggested that the usual varieties of osteoporosis (postmenopausal and Cushing's types) are a mixture of osteoporosis and osteomalacia rather than a pure dietary deficiency of calcium.

Hypercalciuria. It follows from the above discussion that deficiences in calcium can result from excessive and uncontrolled urinary loss. It may well be that the 20% of osteoporotic patients with hypercalciuria are osteoporotic because of their excessive calcium loss.[15] They seem unable to decrease renal wasting of calcium in response to a decreased dietary intake. Normal individuals, on the other hand, can diminish urinary calcium output by 30% after one to two weeks on a low calcium diet.

Malabsorption. Intestinal malabsorption produces a situation identical to deficient dietary calcium. Since dietary fat and steatorrhea are quantitatively related and since vitamin D is a fat-soluble vitamin, malabsorption presumably produces some degree of vitamin D deficiency as well. Lutwak has found a relationship between the quantity of ingested fat and the amount of fecal calcium lost. Conversely, increasing dietary calcium increases the loss of fecal fat.[1]

Isotope tracer studies lend support to the argument that osteoporosis is primarily a disease of increased bone resorption as opposed to de-

creased bone formation. Bone formation rates have been studied in those diseases characterized by alterations in the rate of bone formation. In hyperparathyroidism and osteitis deformans, the rate of bone formation is increased. In myxedema and hypoparathyroidism, the rate of bone formation is decreased. In isotope tracer studies of osteoporosis, the rate of bone formation has been found to be normal, suggesting that decreased bone formation is not the usual mechanism of osteoporosis. In the case of osteoporosis associated with hyperthyroidism, rate of bone formation is known to be increased, but resorption proceeds at a faster rate than formation.

The evidence, therefore, favors a common mechanism of bone atrophy for senile bone disease, endocrinopathy, or simple disuse. While the rate of bone formation may be unchanged or increased, bone resorption is always accelerated in excess of any increase in bone formation.

THERAPY

Osteoporosis not infrequently leads to clinically apparent bone weakness, manifested by bony pain, fractures, muscular weakness, or sensory loss. Bony strength, on the other hand, is related to its mineral content. Vose and associates[17] have shown that osteoporotic bone breaks under a load 40% less than needed to break normal bone.

The therapeutic approach to osteoporosis stems from a consideration of the mechanisms previously discussed. Therapy may be divided into three approaches: definitive care, supportive methods, and "specific" therapy. Definitive treatment means removal of any specific pathological abnormality which is causing the osteoporosis, that is, removal of the parathyroid adenoma, treatment of the hyperthyroidism, or removal of the excessive amount of adrenocortical hormone. Supportive therapy includes those time-honored methods of external bracing and therapeutic exercise to prevent, retard, or correct bony abnormalities. The term "specific" therapy is customarily reserved for the pharmacological approach to the treatment of osteoporosis.

Sex hormones. Reifenstein[18] recently reviewed 18 published balance studies in which patients were treated either with estrogens or with estrogens and androgens. At high doses, significant retention of calcium was seen for thirty days. He therefore recommended the use of sex hormones, claiming a protein and bone anabolic effect without undesirable hormonal side effects upon secondary sex characteristics.

Henneman and Wallach[19] reviewed 200 patients treated for up to twenty years with sex hormones and reported symptomatic relief as well as retardation or cessation of loss of total body height. These authors also reviewed 15 balance studies from the literature and concluded that there was a calcium-retaining effect in 13 osteoporotic patients. It would seem, therefore, that sex hormones, especially estrogens, are probably helpful in producing calcium retention or inhibiting resorption of bone.

Calcium. It appears from the studies mentioned previously that osteoporotic patients are, as a group, eating less calcium than normal individuals. It also seems clear that increased calcium retention can be promoted by increasing the dietary calcium. McCance[20] and Bauer[21] and their co-workers have shown in rats and cats that not only bony strength but also bony structure, as seen on the roentgenogram, can be restored to normal by restoring dietary calcium to normal. Adaptation does occur, however, in patients on a prolonged high calcium intake. Calcium may not be the sole therapeutic approach, but it does seem to play an important role in the treatment of patients with osteoporosis.

Strontium. Although strontium can be shown to be retained after calcium retention has reached a maximum,[22] strontium seems to be of no real value and perhaps may be contraindicated in the treatment of osteoporosis, even though it resembles calcium chemically. Strontium seems to concentrate at the bone surface and may even be preferentially shed with passage of time.[23]

Vitamin D. Osteoporotic patients seem to be deficient in calcium, which may contribute to their osteoporosis. It would seem reasonable, therefore, to treat patients who do not have hypercalcemia or hypercalciuria with small doses of vitamin D. One must remember, however, that vitamin D toxicity can occur from excessive, unsupervised, chronic administration. It should, therefore, be given in relatively small doses (5,000 to 10,000 units per day) with periodic serum calcium determinations.

Protein. Although the malnutrition often seen with osteoporosis would seem to implicate protein deficiency and thereby imply benefit from high protein supplements, balance studies do not show that these patients are, in fact, in negative nitrogen balance.[1] Albright and co-workers,[6] however, did show a reduction in urinary calcium excretion and an increase in serum calcium concentration with intravenous administration of albumin. He postulated that the mechanism may relate to the use of albumin as a transport mechanism for bone precursors.

Fluoride. Fluorapatite has been shown to be more insoluble than the normally hydroxylated apatite.[24] Much experimental work is being done in this field. Several reports to date suggest that

calcium retention can be enhanced by large oral doses of fluoride over a long period of time.[25] The toxic effects of high doses of fluoride, however, must be kept under consideration.

Combined therapy. A broad approach to the treatment of osteoporosis would seem to be appropriate, since no one specific therapeutic measure has been shown to be entirely effective. Until the effect of large doses of calcium in the diet can be investigated further, a diet containing about 2.5 gm. of calcium per day is recommended. Sex hormones may be administered in a balanced and supervised program, together with adequate nutrition and small, carefully supervised doses of vitamin D. It may be that osteoporosis is a disease that affects a heterogeneous group of patients with different primary deficiencies and predispositions.

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Continued from page 117

PRACTICAL REHABILITATION

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orders as traumatic quadriplegia, parkinsonism, multiple sclerosis, chronic pulmonary emphysema, and even metastatic malignant tumors can benefit from attention to their physical rehabilitation. We are increasing our knowledge of the practical aspects of the rehabilitation of hemiplegic, paraplegic, and arthritic patients. With the development of new facilities and techniques, and the increasing availability of trained personnel and economic resources, rehabilitation is now feasible for many patients who would formerly have been neglected or left to their own devices. The physician can now select patients for rehabilitation with more assurance of achieving some degree of success. The general physician maintains a major responsibility for properly guiding these patients, for establishing realistic goals for their rehabilitation, and for communicating these goals and the basic plans of action to the patient and his family.

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Physical Treatment for the Hemiplegic Patient

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Phoenix

THE IMMEDIATE INSTITUTION of procedures to prevent deformities and accelerate physical rehabilitation has an effect on the patient's psychologic acceptance of his disability. This concurs with the fact that physicians who are enthusiastic about rehabilitation have better results than those who apply similar methods in the absence of a hopeful atmosphere.

Eighty percent of stroke victims who survive the medical crisis can learn to walk with or without a cane and a short leg brace. The prognosis for the patient with a paretic arm must be more guarded because of the greater neurologic representation of that extremity in the brain. The secret of success lies in early treatment and adoption of a positive attitude to the end of preventing deformities and maintaining or reinstituting muscle control. When the interest and enthusiasm of the physician and his ancillary staff are ensured, good physical medical treatment of the hemiplegic patient can be maintained in any hospital or nursing home. The techniques are simple and can be mastered with or without the assistance of a physiatrist or physical therapist.

IMMEDIATE MEASURES

The initial phase of physical treatment can be termed "preventive rehabilitation," and it begins concurrently with the onset of disability. This is definitely the most important phase and, if treatment is properly carried out, it can shorten the convalescence and produce better results. Preventive rehabilitation includes use of the following measures:

CVA pack. This pack should be ready when the patient is admitted to the hospital, and it should include the overhead trapeze, bedboards (regardless of the type of mattress to be used), a footboard, and a small rolled towel.

Bed positioning. Proper positioning of the patient is important in the prevention of contractures of the affected extremities (Fig. 1). Unless the position is contraindicated by a cardiac problem, the patient should lie flat, with a small rolled towel placed under the occiput. The affected shoulder should be abducted from the wall of the chest or maintained in an external rotation position to prevent development of a frozen, painful shoulder, a complication which can come about within three days. The forearm and hand should be elevated to prevent edema. The footboard is used to keep the foot in dorsiflexion and to prevent shortening of the Achilles tendon. If the medical condition permits, the patient should be turned to the prone position several times daily to help prevent contractures of the hip and knee.

Immediate institution of passive exercises. These exercises should be carried out through the full range of motion twice daily to the affected limbs.

Stretching of the heel cord. Beginning on the first day of hospitalization, the heel cord on the affected side should be stretched twice daily to prevent shortening of that structure, which, if it is allowed to develop, can become a real problem (Fig. 2).

If the foregoing program is started early and conscientiously, the patient will be well on the way to the restorative phase of rehabilitation.

Performance of rehabilitation within the patient's room should be discouraged; approximately 95% of the patients can be sent to the physical therapy department within forty-eight hours. Such an arrangement serves 3 important purposes. First, it stimulates the patient psychologically and helps to generate a positive approach toward his disability. Second, it allows

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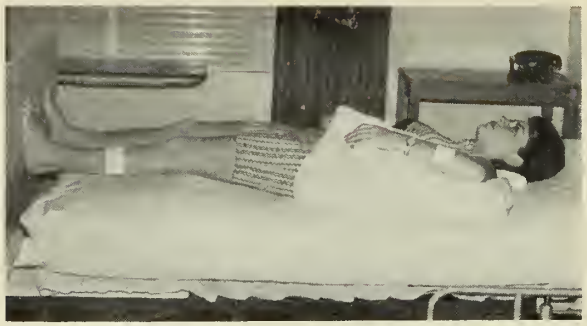


Fig. 1. Proper bed position for a hemiplegic patient



Fig. 2. Correct technique for passive stretching of the heel

better care under direct supervision in the physical therapy department. Finally, it allows members of the nursing service to change mattresses, handle linens, and so on.

RESTORATIVE MEASURES

The restorative phase of rehabilitation often consists of the following program:

Heat. Victims of stroke often have a painful shoulder on the affected side. The use of a heat lamp for thirty minutes plus deep sedative massage alleviates this condition.

Arm sling. By wearing an arm sling to carry the affected upper extremity at an angle of 90° at the elbow, shoulder pain is decreased and edema is prevented. In prescribing the use of an arm sling, the physician must stipulate that the sling carry the weight of the arm across the shoulders instead of across the neck.

Exercise program. When the affected extremities are flaccid, the best exercises to use are passive, relaxed exercises carried out through normal range to all joints of the affected extremities. When some degree of muscle power is regained, this type of exercise can become active-assistive, carried out through the range of motion. The therapist would only assist the power which the patient is able to exert. As soon as possible, reeducation techniques should be started. These consist of teaching the patient the origin, insertion, and function of the muscle involved. This instruction is supplemented by stroking and tapping the affected muscle from the point of insertion toward the site of origin and at times by the use of electrical stimulation to show the patient the function of his paretic muscles.

Ambulation. As soon as the patient is out of immediate medical danger, gait training is started. At first, the tilt table is used in order to stabilize the cardiovascular system. Next, the patient goes to the parallel bars (Fig. 3). He is fitted with a longleg brace affixed to a universal shoe which has a cutout toe or a gutter splint to support the knee and a foot-drop wrap to support the ankle at a 90° angle. When the patient can walk in the parallel bars without support, he is given a cane and he begins to walk outside the parallel bars. The patient should hold the cane in the unaffected hand and should move it forward when the affected leg is placed on the floor.

Transfer activities. Early in the phase of

(Continued on page 129)

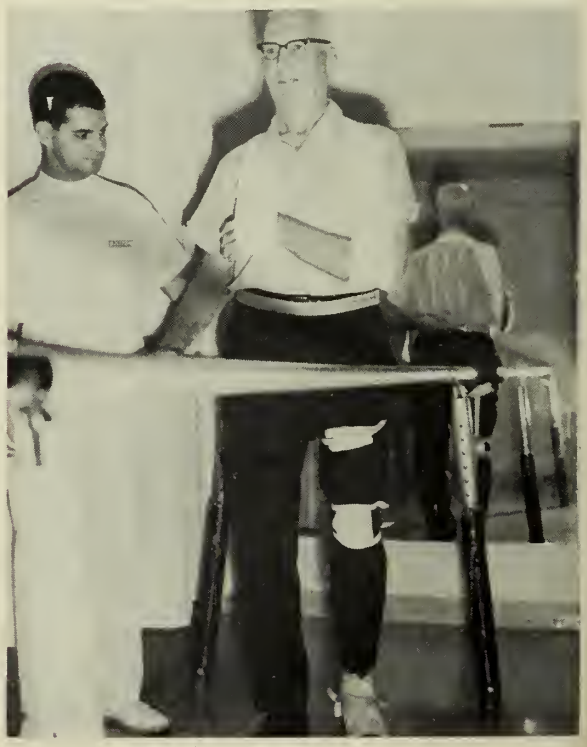


Fig. 3. Balance and gait training of hemiplegic patient in parallel bars. The affected extremities are treated by means of a knee splint, foot-drop support, and arm sling.

Physical Therapy in Obstructive Pulmonary Disease

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VIGOROUS CLAPPING over the back of the chest is commonly done to assist an individual with an inadvertent bronchial aspiration. This is followed by an equally vigorous cough, with expulsion of the offending material. The practice of stimulating a cough by pounding on the chest also is traditionally taught in the care of postoperative patients who develop acute bronchial obstruction.

Largely through the efforts of British workers who have responded to the needs of newly developing thoracic surgery, the role of physical therapy in pulmonary disease has been expanded to the point where programs of breathing exercises and postural drainage have become systematized to deal with a number of specific pulmonary problems.[1–3] The major principle of coordinating the use of gravity for drainage with known anatomical distribution of the segmental bronchi[4] was developed in association with application of physical therapy procedures, such as a vibration-compression maneuver and clapping over the chest.

More recently, interest has developed to utilize effective procedures to relieve the bronchial obstruction by an abnormally viscous mucus in patients with cystic fibrosis. The basic principle for such procedures is that, in any biologic ductal system, restoration of optimal physiologic functioning can be achieved only with removal of the obstruction. Since it has been realized that antibiotics alone are inadequate in the treatment of bronchial obstructive disease of any kind, this interest has broadened to include both children and adults with such conditions as chronic bronchitis, agammaglobulinemia, bronchiectasis, and postoperative pulmonary atelectasis. In addition, the procedure has been found useful in acute paralytic disorders involving impaired respiratory muscular function, such as severe Guillain-Barré syndrome, where pulmonary complications are common.

Experience with the disease, cystic fibrosis, has shown that a consistent program of bronchial drainage in conjunction with other measures, such as vitamins, antibiotics, aerosol mist, and digestive enzymes, has decreased the severity of pulmonary involvement in these children. Doershuk and co-workers'[5] review of a five-year, multifaceted program has shown improvement in 82% of patients treated and a decline in mortality to 3% from estimates as high as 15%.[6] Those in the latter category were children who had begun the program at a relatively far-advanced stage of the disease. In contrast to their previous experience[5] where the death rate of children under 5 was 25%, no children under 5 died in this series.

To be effective, a program of bronchial drainage must originate with a physician who is personally convinced of its value. This conviction must be transmitted to the patient and the family in order for them to devote the time and effort necessary for regular daily treatment. The physician should have a working knowledge of the segmental anatomy of the lung in order to correlate physical and X-ray findings with the treatment to be prescribed. In the chronic lung diseases, repeated evaluation is necessary to determine at any one time the localization of the obstructed segments.

The bronchial drainage procedure consists of the application of 3 basic physical therapeutic maneuvers designed to mobilize mucus and secretions within the bronchi and to stimulate coughing. These procedures are: [1] manual vibration and compression of the thorax, [2] clapping the chest wall, and [3] positioning to facilitate gravitational drainage from segmental bronchi.

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These procedures are coordinated to correspond with the particular bronchopulmonary segment to be treated. The patient is placed in a position which will allow the bronchus draining the area to be oriented in a vertical direction. The area of the chest over the respective pulmonary segment is treated by manual vibration compression and clapping. When involvement is generalized, the regular sequence of positions provides systematic drainage of all segments and insures that no segments will be neglected.

MANUAL VIBRATION COMPRESSION

In the application of vibration compression, the hands are placed on the chest over the area of the segment to be treated. Usually one hand is placed on top of the other, the hand in contact with the chest being curved to conform to the configuration of the chest wall. Isolated pressure of the fingertips or finger joints usually produces discomfort and should be avoided. The patient's respiration is observed, and vibration and compression are applied during expiration only. Where respiration is rapid, as in infants or severely ill patients, compression vibration is applied during every second or third expiration. Both hands are caused to vibrate in a direction perpendicular to the skin's surface. Movement of the chest wall in response to vibration should go through an excursion of about a half inch in infants and small babies to an inch or more in older children and adults. In the apical region of the chest anteriorly and the entire upper portion of the chest posteriorly, the range of movement will be considerably less than this. The force to be applied should be proportionate to the size of the child. Pain or severe discomfort, overfatigue, and interference with inspiratory efforts should be avoided. A rate of 6 to 8 vibrations per second is generally satisfactory and appears to produce effective coughing. With the vibration, a compressive force is applied (most easily with the uppermost hand) during the entire expiratory phase of respiration.

CLAPPING

Alternating with vibration compression, clapping is applied to the chest wall over the segmental area being treated. Both hands are used to achieve a 6- to 8-per-second beat similar to that in compression. A towel or the clothing is used to prevent smarting during the clapping. The hands and wrists are held relaxed and are curved to conform to the chest contours. Alternatively, the hand may be cupped and, if correctly done, the cushion of air that is trapped provides protection against painful slapping.

POSITIONING

There are 13 positions relating to drainage of the major bronchopulmonary segments. Each position is calculated to place the segmental bronchus in as nearly a vertical position as possible. They are achieved by use of a table or board which can be placed at a convenient working height and can be tilted at various angles. For convenience in achieving some of the positions, 1 or 2 pillows are used, either to stabilize the patient or to enhance the angle of tilt. The 3 angles at which the table is placed, corresponding roughly to the upper, middle, and lower lobes, include: [1] horizontal, [2] foot elevated 12 in., and [3] foot elevated 18 in.

The sequence of positions is presented in such a manner that at each angle the patient goes through a single rotation, stopping at the appropriate points for specific segments. Each position is maintained three to five minutes, but the time may be modified.

PROCEDURES WITH TABLE HORIZONTAL

1. *Apical bronchus of the right upper lobe.* In this position, the patient sits upright, with legs dangling (Fig. 1). Support is given to the patient's back by leaning against it or pressing with one hand while the other hand applies the



Fig. 1. Position relating to the apical bronchus, with the angle corresponding to the right upper lobe



Fig. 2. Position relating to the anterior bronchi, with the angle corresponding to the right and left upper lobes

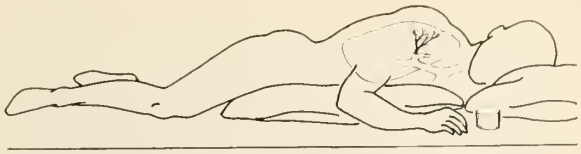


Fig. 3. Position relating to the posterior bronchus, with the angle corresponding to the right upper lobe

vibration compression to the infraclavicular area on the right side. During the treatment, the patient leans backward for short periods of time, then forward, and then sideways to right and to left in order to drain the entire segment. If coughing begins during any part of the therapy, the procedure is interrupted and the patient's coughing is assisted by supporting the lower costal and upper abdominal area with the hands. Coughing is encouraged at all times.

2. *Anterior bronchi of the right and left upper lobes.* In this position, the patient lies on the back (Fig. 2). Pillows may be placed under the knees and the head for comfort. The upper anterior portion of the chest at the level of the second to fourth ribs overlies approximately the segmental area to be treated. Vibration compression and clapping may be carried out over both right and left sides.

3. *Posterior bronchus of the right upper lobe.* From the back, the patient turns to the left side with the right side of the body turned forward to an angle of about 45° (Fig. 3). A pillow is placed in the left or lower armpit and under the left chest for support. In this manner, the posterior segment of the right upper lobe is brought into the highest position and the bronchus directed most nearly vertically. Clapping and vibration compression are applied to the upper posterior thorax over the area of the lateral angle of the scapula.

4. *Superior bronchi of the right and left lower lobes.* The patient lies prone, with a pillow placed under the abdomen in order to flex the trunk slightly (Fig. 4). The treatment is applied to the back of the chest bilaterally at the level of the lower angle of the scapula.

5. *Apical posterior bronchus of the left upper lobe.* The patient lies on the right side, with the left side of the body turned forward at an angle of about 45°. A pillow is placed in the right axilla and under the right chest for support. This position is the exact counterpart of the position for the posterior bronchus of the right upper lobe, and therefore no diagram is shown.

PROCEDURES WITH FOOT OF TABLE ELEVATED 12 IN.– LOWER DIVISION, LEFT UPPER LOBE (LINGULAR)

The foot of the table is elevated 12 in. for the next 2 positions. The patient lies on the back, with the body turned toward the right at an angle of about 45°. The back is supported with a pillow which extends from the left shoulder to the left hip. Treatment is applied over the lateral anterior portion of the left chest from about the level of the third to the sixth ribs. This position is comparable to that relating to the right middle lobe.

6. *Right middle lobe.* The patient lies on the back, with the body turned toward the left side at an angle of about 45° (Fig. 5). A pillow under the back extends from the right shoulder to the right hip for support. The compression vibration and clapping are applied to the right anterolateral portion of the chest from the third to the sixth ribs.

PROCERUDES WITH FOOT OF TABLE ELEVATED 18 IN.

For the remaining positions, the foot of the table is elevated 18 in. To treat each area systematically, the patient makes a complete rotation during the treatment, stopping at this level at 6 positions corresponding to each of the major segmental bronchi of the lower lobes.

7. *Medial basal bronchus of the right lower lobe.* The patient lies on his back (Fig. 6). Compression vibration and clapping are applied over the lower portion of the right anterior rib cage.

8. *Anterior basal bronchus right lower lobe.* This bronchus is slightly lateral to the medial bronchus, so the patient turns slightly to the left. Treatment is applied slightly laterally.

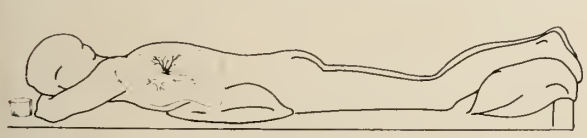


Fig. 4. Position relating to the superior bronchi, with angle corresponding to the right and left lower lobes

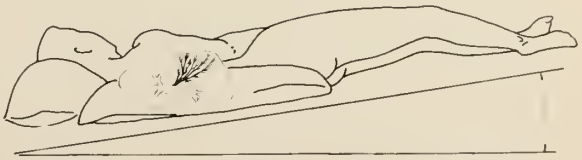


Fig. 5. Position with the foot of the table elevated 12 in., corresponding to the right middle lobe

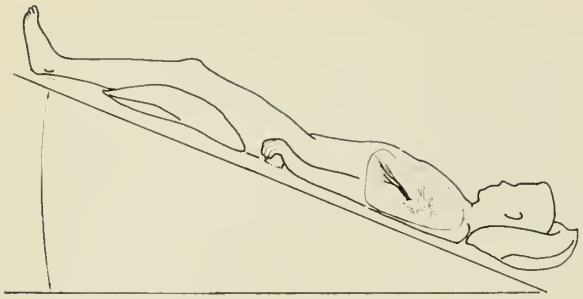


Fig. 6. With foot of the table elevated 18 in., position relates to medial basal bronchus of the right lower lobe.

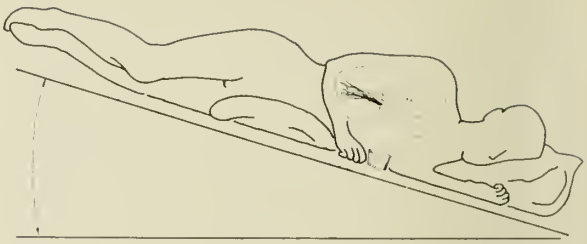


Fig. 7. With foot of table elevated 18 in., position relates to the lateral basal bronchus of the right lower lobe.

9. *Lateral basal bronchus of the right lower lobe.* The location is still more lateral, and the patient lies directly on the left side with the pillow under the crest of the ilium and loin to increase the angle of tilt (Fig. 7). The patient should be observed in this position, as in the others, to make sure that pillow placement effects a downward tilt of the thorax. If the pillow is placed too close to the trochanters, the lower chest will sag and the inferior portions of the basal bronchi will actually be lower in level than some of the more proximal regions. Treatment is applied to the right lower chest over the lateral surface.

10. *Posterior basal bronchi of the right and left lower lobes.* In youngsters, this position is most easily achieved by placing the hips on 2 stacked pillows so that the thoracic spine is positioned at an angle of 45° to the floor (Fig. 8). Treatment is applied to the lower chest posteriorly and bilaterally. The same position can be achieved in older children and adults by having the individual hang over the edge of the bed with arms on a chair or the floor. This is the position commonly associated with the term "postural drainage" and has been used in adults frequently for the treatment of chronic bronchial disease, such as bronchiectasis. However, since emphasis in this treatment is placed equally on the vibration compression and clapping as well as the positioning, the preferred term is "bronchial drainage."

11. *Lateral basal bronchus of the left lower lobe.* The patient lies on the right side and a pillow is placed under the right lower ribs and ilium. Treatment is applied to the lower lateral area of the left rib cage. The position and area to be treated are a mirror image of the right side, and the diagram for the right lateral basal bronchus may be referred to for this position.

12. *Anterior medial basal bronchus of the left lower lobe.* The patient lies on the back, with the body turned slightly onto the right side. A pillow along and underneath the back on the left will provide necessary support and prevent rolling. Treatment is applied to the lower anterior portion of the left chest.

DISCUSSION

When a young infant is to be treated, each of these positions can be modified by placement on the lap. Tilting can be accomplished by either crossing one's legs and laying the child across the lap in the appropriate direction or by placing the feet on a footstool. When the baby is tilted head downward, a firm grasp of the lower extremities by the elbow and forearm of the individual treating the child will prevent slipping. When respiration in a young infant or child is extremely rapid, it may be necessary to apply the compression vibration during every second or third expiration. It is essential that no compression be applied during inspiration, as this fatigues the patient.

Emphasis should be placed on allowing adequate time for this procedure. Each position should be maintained at least three to five minutes, depending upon the amount of mucus produced. When physical findings are particularly striking and localized, the time of treatment and the frequency of repetition of treatment to a particular area may be increased to three, four, or more times daily. When involvement is local-

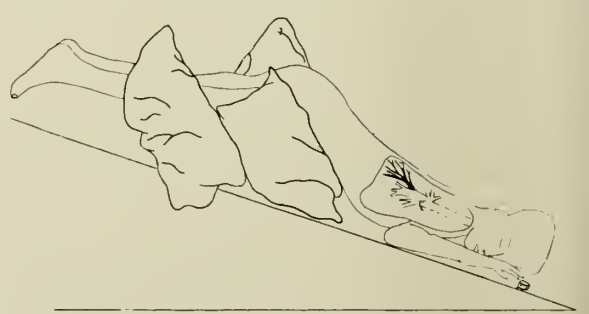


Fig. 8. With foot of the table raised 18 in., the position relates to the posterior basal bronchi of right and left lower lobes.

ized to a few segments, treatment may be concentrated to these areas only. If follow-up is close, the prescription may vary from time to time depending upon the degree and location of involvement. Most children with cystic fibrosis, however, have fairly generalized involvement, particularly if treatment has been inadequate. When the chest is consistently clear, it has been felt necessary on empirical grounds (in the case of the child with cystic fibrosis) to continue treating a number of selected areas. This might include the upper apical segments utilizing the first position for both sides, the right middle lobe, the right and left lateral basal segments, and the right and left posterior basal segments. The freedom with which the treatment may be discontinued completely should depend on the rate at which mucus collects in the bronchus as observed on previous experience, as well as the practicable frequency of examination. In any case, patients or their families should be instructed to resume treatment with onset of respiratory infection or spontaneous coughing. Where the etiology is another condition, treatment may be interrupted completely if judged advisable.

The clinical condition of the individual will dictate the extent of treatment which can be carried out in any one session. For patients with limited activity tolerance, dyspnea, or right heart enlargement, a full treatment program for one hour often will be exhausting, if not hazardous. Treatment should be observed and, if necessary, 1 or 2 segments may be treated at spaced intervals during the day. In general, it is wise to have at least 2 drainage treatments daily for each segment—and more if a particular segment is severely involved. Treatment is best given in the morning directly on arising and immediately after inhalation of aerosol mist. In the evening, the treatment is best given at least one hour after meals to avoid vomiting.

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Continued from page 124

PHYSICAL TREATMENT FOR THE HEMIPLEGIC PATIENT

Carl R. Bjorklund, M.D.

restorative rehabilitation, the patient can be taught to transfer himself from the bed to the wheelchair, from the wheelchair to the bed, to and from the toilet, and to and from an automobile. Patients accomplish these transfer activities easily by using the unaffected arm and leg for support and pivoting.

Bracing. When paresis of the lower leg persists, the proper appliance is a shortleg, double upright brace fitted with a 90° stop at the ankle. Such a stop prevents plantar flexion, an important consideration in the prevention of strengthening of the gastrocnemius muscle over the anterior tibialis. A spring wire brace or an ordinary Klenzak brace can strengthen the gastrocnemius and soleus muscles to such a degree as to accentuate the extensor thrust of the affected extremity. Often, the shortleg brace is a temporary measure. If function returns in the dorsiflexor muscles of the foot and ankle, the brace can be discarded.

COMMENT

Victims of stroke generally are ambulatory, in one form or another, within a week to ten days, and almost all who have sustained cerebrovascular accidents can return home within four to six weeks. This does not mean that complete function is regained, especially in the upper extremity. Rather, ambulation and self-sufficiency in the management of the patient's needs and wants are the primary objectives. When the economic factors of insurance, the shortage of beds, and the need for family cooperation are taken into consideration, this program will be found quite satisfactory. The longer patients lie in bed, the less dramatic and effective the results of treatment will be, and often the attainment of realistic goals will be thwarted.

Presented at a symposium on Upgrading Physical Medicine Services in the Community Hospital before the Section on Physical Medicine of the American Medical Association, June 22, 1964, San Francisco

Transfers and Mobility for Paraplegic and Quadriplegic Patients

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In the past two decades the number of patients who have survived spinal cord trauma with a residual paraplegia or quadriplegia has increased significantly. Increasing numbers of accidents, chiefly automobile accidents, are creating the condition while steadily improving treatment of the acute trauma saves lives that a few years ago would have been lost. Thereafter, because of improved methods of maintenance, these severely disabled people can look forward to a life-span not greatly reduced from that of the general population. The majority of the cord-injured are young people who were in good health at the time they were injured. Thus they face several decades of living with a severe disability.

In our present state of knowledge, repair of the damage is not possible. No medicine, no surgery, no physical agent, no massage or any other therapeutic effort will restore the disrupted neural mechanism. What then can we as physicians have as goals of treatment for a person in whom the lower part of the body is no longer under neurological control? The pertinent goals can be summed up in the following manner: (1) to help the patient adjust his value system so that he can accept himself as a worthwhile person in spite of the functional impairment, (2) to help him learn how to tend to his own needs as far as is possible within the limits of his disability, so that his dependence on others is minimal, and (3) to teach him to take care of the neurologically isolated part so that it does not deteriorate and destroy him. The present paper is concerned with the second goal, that of increasing one aspect of independence—mobility, being able to get from one place to another.

Independence in mobility is basically composed of the successful accomplishment of many small activities, including such simple maneuvers as getting from a bed to a chair and back again. The amount of independent mobility possible depends in large part on the level at which the lesion occurs. The lower the level of injury to the spinal cord the greater will be the residual voluntary neuromuscular control and the degree of independence that the patient can achieve.

The most frequent site of injury in the neck just spares the sixth cervical segment of the spinal cord, which leaves the patient without elbow extension and without hand function. Transferring ability depends on the ability to keep the elbow extended, which usually requires at least an intact C_7 segment to supply the major portion of the triceps, and the reverse use of the shoulder depressors, particularly latissimus dorsi (with major innervation C_6, C_7, C_8) to raise the body.

Ability to transfer is also somewhat dependent on individual variability, including such factors as body build, agility, and motivation. As an illustration, a 19-year-old lad with a C_{5-6} quadriplegia with exceedingly weak wrist extension, no elbow extension, and only strong biceps and shoulder muscles to work with is able to transfer independently from bed to wheelchair and back again and to transfer in and out of a car with very little assistance. This young man happens to be of an athletic tendency, has no tightness, and is highly motivated.

Most quadriplegics, because of difficulties with balance coupled with weak shoulder depressors and absence of hand function, cannot transfer independently. Even if some hand function is present the patient may have inadequate balance to transfer without assistance. However, there are devices that minimize their dependence. The simplest and most portable method is the use of a sliding board and a transfer belt, by which one person can slide the patient to the desired

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location without lifting him (Fig. 1). The assistant places the sliding board in the proper position and then helps the patient in the actual mechanical task of sliding across the board. A transfer belt allows this to be accomplished without great muscular effort on the part of the helper. It is important, of course, that the chair and bed be at approximately the same height so that the sliding board is horizontal. Also it is essential that the wheelchair be equipped with brakes and that the footrests can be swung out of the way in order to provide room for the legs. A removable armrest on the side on which the transfer is being made is also needed; the armrest may be hung on the opposite handle of the wheelchair during the transfer so that it is out of the way, and the patient can often assist by using the upper extremity against the armrest that is still in place.

A patient must be able to sit up in bed before he can transfer. Often a person quadriplegic at the C_6 level can accomplish this only by using partial side rails on the bed, as shown in Figure 1. He can then use his intact biceps and shoulder muscles to pull himself to a sitting position.

There are some patients who prefer a direct anterior-posterior sliding transfer, which is illustrated in Figure 2. This requires loose hamstrings and slightly more strength, particularly of the elbow extensors, than the lateral transfer. These conditions are perhaps best met by the flaccid paraplegic.

On the other hand, a paraplegic individual who has normal upper extremities and at least a partially innervated trunk should be capable of independent transfer to and from bed, requiring no side rails, and furthermore, he should be able to transfer into a car and pull the wheelchair in after him. This is usually best accomplished in a 2-door car, where the wider door and the lack of a post in the middle combine to make easier access to the space behind the front seat. Transferring directly in this manner does not usually require a sliding board and many paraplegics prefer fixed armrest on the wheelchair, particularly if they are muscular and can forcefully raise themselves up and swing over to the desired location.

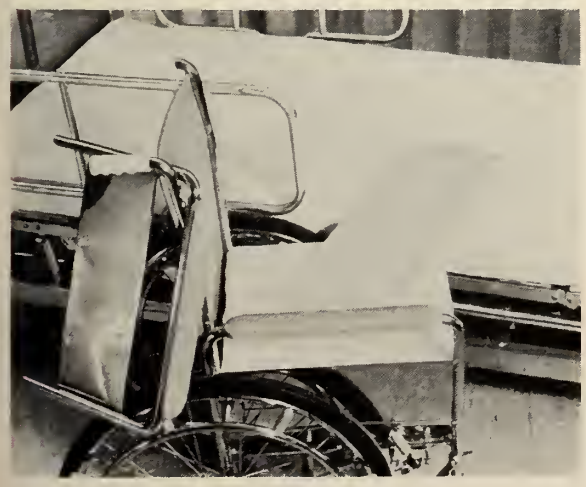


Fig. 1. Equipment readied for sliding board transfer



Fig. 2. Direct anterior-posterior sliding transfer

Paraplegics whose level of injury is below T_{12} often are able to walk with long leg braces and crutches and can transfer from sitting to standing without assistance.

In any of these cases the patient (who usually has analgesia in the paralyzed parts) should be thoroughly impressed with the need to avoid striking metal parts of the chair or other objects that could cause abrasions.

Some cord-injured persons, particularly those who will be mainly confined to their homes, prefer a trapeze bar to aid in transferring (Fig. 3). For efficiency the bar should be suspended from 2 separate points rather than only 1, as is commonly done. The bar should be long enough to extend slightly beyond the side of the bed. It is usually suspended from an overhead frame attached to extensions built up from the bedposts.

Besides bed-to-chair and chair-to-car transfers, one of the most important transfer abilities, particularly for the paraplegic, is the toilet transfer. Here again the question of height is important. In most instances the use of a raised toilet seat which is firmly attached will permit the level of the chair cushion to be approximately the same as that of the toilet seat (Fig. 4). In the average home it is most unusual to find the bathroom fixtures arranged so as to make the side-to-side transfer technique possible. Therefore, even in the best of circumstances one would have to maneuver from the side of the

wheelchair to the front of the toilet. Then for proper balance 1 or 2 hand grips are needed, firmly anchored on the wall (not like most towel racks), to assist in the transfer.

Of course, the toilet transfer is of little practical value to the man whose bathroom is so small that it will not accommodate the wheelchair at all. In these circumstances a commode can be used. One of our former patients has even devised a portable, collapsible commode which makes it possible for him to travel around the country and stay in any motel he pleases without regard for the arrangement of the bathroom or the size of the door. He is now marketing this device. The armrest is removed from one side of the chair and two short posts of the commode fit in where the side rail armrests were located, so that he can transfer onto his commode. He uses a plastic bag, obtainable in any grocery store, for collection of the wastes and then deposits it in any nearby trash repository.

Tub transfers for the paraplegic usually require a straight chair next to the tub and firm





Fig. 3. Overhead bar transfer

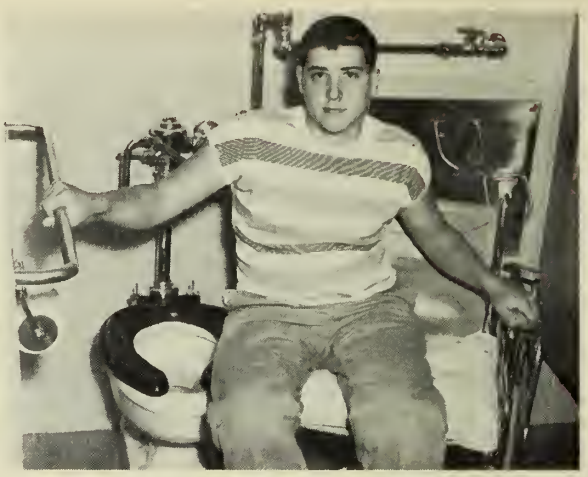


Fig. 4. Wheelchair-to-toilet transfer

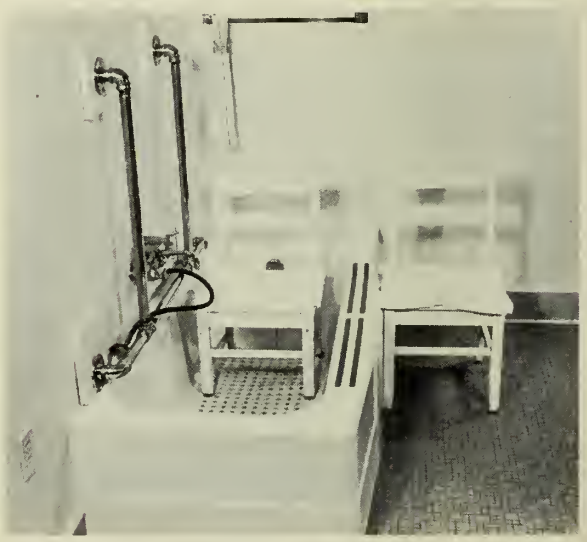


Fig. 5. Chair-to-chair tub transfer

hand grips on the wall at the side of the tub for easing down into the water. Strips of nonabrasive adhesive material that increase friction are very helpful on the flat part of the side of the tub, especially for getting out, since the wet porcelain is very slippery (Fig. 5). Some patients prefer another chair in the tub at a lower level so that they do not have to raise and lower their bodies so far. Others use this arrangement when there is an attached shower. Patients whose tubs are equipped with sliding glass doors may find that a loop attached to the upper frame helps them to transfer both in and out of the tub.

Another mechanism that can be very useful for increasing mobility is the hydraulic mechanical lifting device for helping a patient in and out of bed. A similar device can be applied to the roof of a car to help a person in and out, and there are further variations to help patients get

(Continued on page 139)

Physical Medicine for Patients with Rheumatic Disease

DOROTHY M. STILLWELL, M.D.

Denver

THE MANAGEMENT of a patient with arthritis is complex and of long duration. Early in the course of rheumatic disease the patient generally complains to the physician of pain in a joint. Examination shows that the muscles around this joint are in spasm. This "splinting" protects the joint to some degree, but also increases substantially the degree of pain which the patient experiences. Treatment should be instituted at this time, rather than later. If the pain continues in a joint, muscle weakness will develop, partly because of reflex inhibition leading to atrophy and partly because of disuse. In both instances the stimulus is pain.

When the triad of pain, muscle spasm, and weakness is present, deformities may develop. In some cases of rheumatoid arthritis, crippling progresses in spite of early treatment, but this is not a valid reason for not treating each patient from the onset of the disease. If physical measures are to be effective for such a patient, it is necessary not only to treat the signs and symptoms but also to consider future problems which may arise, and to make every effort to prevent them. At all times the program should maintain an efficacious balance between adequate rest and activity of the joints.

EARLY RHEUMATOID ARTHRITIS

Acute. For the patient who has acute rheumatoid arthritis rest in bed may be the treatment of choice, particularly when numerous joints are involved. At such a time the use of physical modalities may be limited to correct positioning of the bed to prevent deformities. If necessary, plaster shells can be used to maintain the knees and wrists in good position. Otherwise, correct positioning requires adequate use of a footboard, sand bags, and a small head pillow to achieve good anatomic alignment. Actually, such positioning should be used regardless of the stage of the disease.

Subacute. Once the patient's acute illness is subsiding, physical measures can be used more actively. The application of heat in the form of hydrotherapy seems to be particularly advantageous. In a department of physical medicine and rehabilitation the Hubbard tank or the standard large whirlpool bath can be used. A water temperature of between 100 and 103° F. is used, depending on the heat tolerance of the patient and the extent of the body to be heated. Treatment generally lasts twenty to thirty minutes. Pain is reduced by the direct effect of heat and also indirectly by the relaxation of muscle spasm.

Methods of positioning and splinting to prevent deformities should be continued during the subacute phase.

When pain and muscle spasm decrease, a program of exercises is employed to maintain or increase range of motion. These exercises should be given to all joints of all extremities at least once daily. They can be carried out while the patient is receiving hydrotherapy, or they can follow heating of the part.

Since muscular weakness also is a component of rheumatoid arthritis, an additional program of strengthening exercises must be employed. Frequently the patient will complain of pain if isotonic strengthening exercises are used. If the patient is following an adequate program of range of motion exercise, isometric exercises may be used to strengthen the muscles. Since isometric exercises do not require the movement of a joint, the patient is more likely to attempt them because they rarely increase the pain. These exercises are difficult for some patients to perform. Initially, the program should be supervised closely by a physical therapist. Unless maximal muscle tension is developed and held for a reasonable length of time, strengthening will not be achieved. The patient must comprehend this basic physiological principle.

By the time the patient is discharged from the

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hospital, a home treatment program should have been well established. The patient should continue to apply heat regularly at home; for example, a heat lamp, heating pad, hot-water bottle, tub of warm water, hot shower, paraffin for the hands and wrists, or contrast baths. The patient probably will have been instructed to observe a rest period each day. This is the ideal time for the application of heat followed by the performance of exercises. Many persons who have rheumatoid arthritis are young housewives with small children and they are encouraged to carry out their physical treatment programs while the children are napping.

There are 2 occasions when a patient is tempted to discontinue a home exercise program. The first is when he has no symptoms and the second is when there is an exacerbation of symptoms. The first instance requires adequate education of the patient while the second obviously requires medical attention.

The patient's daily activities should be evaluated carefully. Many household activities can be altered to prevent overactivity of certain joints. A high kitchen stool, for example, can be used while the housewife is washing dishes or preparing meals, and by sitting during these periods she effectively rests the weight-bearing joints. Many other minor and inexpensive alterations can be instituted to reduce overuse of involved joints.

Late stages. When a rheumatoid arthritic patient is first seen in the late stages of the disease, with deformities already present, the problem of treatment is much more complicated. There is no way in which physical measures can alter the destructive lesions of joints and many of the deformities which have developed.

Since orthopedic surgical treatment is now being employed for these patients with increasing frequency, physiatrists and general physicians are encountering many more preoperative and postoperative rheumatoid arthritic patients. If the surgical procedure is such that the patient will require crutches postoperatively, it will be much easier to teach the patient his appropriate crutch gait preoperatively. In our experience preoperative instruction of this type has effectively reduced the length of hospitalization.

Since the operative treatment is likely to correct limited range of motion, it is important that an exercise program be well established preoperatively so that the patient will be capable of performing these exercises postoperatively, despite the additional pain at the surgical site. While the patient is in the hospital, it should always be remembered that the other extremities should be exercised to maintain available range of motion and to keep muscular strength at its preoperative level. Both physical therapy and occupational therapy can be used effectively for this maintenance program.

OSTEOARTHRITIS

Early stages. The patient who has osteoarthritis with pain in a joint also has muscle spasm. Frequently the joint involved is a weight-bearing joint. As is true of the patient with rheumatoid arthritis, it is important to train this patient early in maintenance of careful balance between rest and activity of this joint. Often it is necessary to prescribe crutches and teach the patient a nonweight-bearing gait for the involved extremity so that the painful joint will be rested sufficiently. Indeed, if the use of a nonweight-bearing gait with crutches does not eliminate the pain, it may be necessary to prescribe complete bed rest until the pain has subsided.

Heat is beneficial in the early stages of osteoarthritis. If at all possible, forms of heat which the patient may apply at home should be used. This would include superficial forms of heat such as hydrotherapy or hotpacks. If the hands are involved, contrast baths or a paraffin bath can be used. All of these modalities can be applied by the patient in his home. When the patient has been well trained in therapeutic measures to control his symptoms, a program can be established quickly at home. Range of motion and strengthening exercises are once again important parts of this program.

Late stages. The patient with advanced osteoarthritis is treated in much the same way. The patient who has advanced disease, however, is more likely to have muscular weakness as well as spasm about the involved joint. It will be necessary, therefore, to increase the time spent on an exercise program to increase the strength of musculature about the involved joint.

Frequently we see patients with advanced osteoarthritis before surgical treatment of a diseased joint. All previous comments about the preoperative treatment of rheumatoid arthritic patients apply to the osteoarthritic patients as well. In the late stage of osteoarthritis one is likely to encounter bilateral involvement of weight-bearing joints. Ambulation then becomes a greater problem. Fortunately, it is unusual to encounter a patient whose pain is of equal degree bilaterally. Crutches are advisable. It is necessary to regulate the length of time the patient spends in nonweight-bearing, or in partial weight-bearing on the involved side in order to avoid an exacerbation of symptoms on the oppo-

site side. When involvement and severity of symptoms are equal bilaterally, the patient must be taught to use his crutches in such a way that the extremities are assured an equal period of rest. Indeed, in such a case complete bed rest may be the only way in which to give effective rest to these joints.

OSTEOARTHRITIS OF THE CERVICAL AREA OF THE SPINAL COLUMN

Osteoarthritis of the cervical spine is fairly common, but treatment of a patient with this particular form of the disease is slightly different. Such a patient responds well to the application of traction. Fortunately, traction sets are available commercially at a reasonable cost for use in the home. An effective routine for these patients is to give them a course of 6 to 12 treatments within the department of physical medicine and rehabilitation. Usually we see these patients 3 times a week. Treatment includes the application of heat, followed by massage, which in turn is followed by traction. It is important that the traction be exerted in the midline, without flexion or rotation of the cervical spine.[1] We find that most patients will certainly tolerate at least 16 lb. of traction and in most instances traction can be gradually increased to 20 or 25 lb. Once the patient has responded to such a course of treatment, a program is immediately instituted at home.

In addition to the use of the foregoing modalities, these patients wear a felt or sponge-rubber collar. The patient is instructed to wear this collar particularly at night. If the patient has symptoms during the day he is encouraged to wear the collar constantly. Nocturnal wearing of a collar is based on the assumption that once asleep, the individual has much less control over the degree of movement in the cervical spine. In the histories of such patients it is common to find notations that they have been awakened during the night by pain or that discomfort is greatest in the morning when they awaken. When osteoarthritis in this region is severe and refractory to this general outline of treatment, a more extensive neck brace may be required, such as a four-post cervical brace. Such a brace provides greater immobilization of the cervical spine. Most patients have difficulty adjusting to the use of such a collar and when they first try to wear it they require much encouragement from their physician. A four-post collar is uncomfortable to wear during sleep and hence the patient will require a simpler device at night.

COMMENT

It is of the utmost importance that both the rheumatoid arthritic patient and the osteoarthritic patient be well schooled about his disease in the same manner in which a diabetic person is taught the salient features of diabetes. The arthritic patient must assume his share of responsibility for his treatment program. If this understanding is not established early, the patient will develop the attitude that such a program can be done, and in fact should be done, only by a physical therapist. Such a patient will not carry out a program at home. Conversely, when a patient has been adequately instructed and advised, he will pursue a program at home which may be satisfactory for long periods of time. If a flare-up in a joint is so severe that it does not respond to the measures of a home program, the patient most certainly deserves more extensive treatment, which can be administered in a department of physical medicine and rehabilitation. In view of the number of arthritic persons in any community, the importance of the patient's assuming responsibility for part of his treatment is obvious. Therapists can then devote their time to treating the patient who is unable to manage the situation effectively at home.

Presented at a symposium on Upgrading Physical Medicine Services in the Community Hospital before the Section on Physical Medicine of the American Medical Association, June 22, 1964, San Francisco, California.

REFERENCE

1. GREEN, DAVID, and JOYNT, R. J.: Vascular accidents to the brain stem associated with neck manipulation. J. Amer. med. Ass. 170:522, 1959.

The Early History of Physical Medicine and Rehabilitation at the University of Minnesota

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Minneapolis

THE EARLIEST use of physical modalities at the University of Minnesota of which I am aware, was the employment of conventional diathermy in the student health service. Doctor Harold S. Diehl was at that time on the health service staff and became interested in the use of this modality in medical treatment. Mr. Ray Amberg was then a pharmacist and served as the technician for application of this treatment.

The fact that Doctor Diehl later became dean of the medical school and that Mr. Amberg became hospital administrator was of great value when the time came to establish a complete department of physical medicine and rehabilitation, because of their interest and active support.

When the Elliot Memorial Hospital began service in 1911, there were no facilities for physical therapy. In 1928, when the Eustis Hospital was built, a small physical therapy department was included. This department was located on the first floor across the hall from Eustis Amphitheater. It consisted of 3 rooms, 1 of which was occupied by a very inadequate therapeutic pool. This pool was approximately 10 ft. long by 6 ft. wide so that it was not large enough for the therapist to get into the water with the patient but was too large for the therapist to lean over the edge to administer treatment. Of the other rooms, 1 contained a few treatment booths and the other served as a small gymnasium.

I have been told that the earliest treatments were given by Mr. Ewald Chalberg, a Swedish masseur who maintained an office in the Medical Arts Building in Minneapolis, but came into the hospital on a part-time basis to treat patients. Whether he received his remuneration from the hospital, the patient, or gave his services as a charitable contribution, I do not know.

Doctor Wallace H. Cole, the chief of orthopedics, had unsuccessfully tried for some time to get a full-time physical therapist, but after a mild poliomyelitis epidemic in 1931, a Mr. Savage who had 4 sons afflicted with the disease, one of them severely paralyzed and in a respirator, put enough pressure on the administration to obtain one.

Miss Sara Kollman came from Gillette Hospital to the university on Jan. 1, 1932, to give massage, exercises, and pool treatments under Doctor Cole's direction. Stall bars, weights and pulleys, posture mirror, and other equipment which had been stored in the unused therapeutic pool room were installed and the department began to function. Gradually, patients began to be referred by medicine and neurology as well as by orthopedics. A limited number of outpatients also received treatment.

However, the department was divided. Doctor Wilhelm K. Stenstrom, a biophysicist in charge of X-ray therapy was in charge of electrotherapy. Bernice Harlow Swanson and later Laura Becklund Plank, nurses from the X-ray department gave diathermy and ultraviolet treatment 3 times a week.

At least 3 clinical research projects were studied at this time. Treatment of peripheral vascular disease by suction-pressure boots and cuffs was investigated by the surgical service; Doctor Flagstad, an orthopedist, studied the effect of scoliosis on vital capacity; and intensive mechanical fever therapy using the Kettering Hypertherm was investigated by Doctor Malcolm M. Cook.

During my time as a medical student (1924-1930) the only mention of physical therapy was contained in 2 lectures given by Doctor Sten-

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strom. These were principally on the physics of diathermy and radiant energy. He was an excellent man and as I grew to know him later I had great respect and admiration for his ability and knowledge and his kindly attitude. However, these lectures were delivered with a pronounced Swedish accent, were not clinically oriented, and made little impression upon me or the other members of my class. They were given in the sophomore year when we were all anxious to begin clinical work and learn clinical things and were totally disinterested in basic sciences.

My own interest in physical therapy stems from my concern about adequate treatment and restoration of function in fractures during my surgical residency at the Minneapolis General Hospital from 1930 through 1933. Upon completion of my surgical residency, I was appointed chief of the department of physical therapy because of this interest. Realizing that I knew almost nothing about the field I made arrangements to spend two weeks at Michael Reese Hospital in Chicago with Doctor Charles Molander. I also accompanied Doctor John F. Coulter on his rounds of several of the hospitals in Chicago. There was no such thing as a residency in physical therapy in the United States at that time.

I then returned to Minneapolis and went into general practice. I found a good deal of free time to devote to the department of physical therapy at Minneapolis General Hospital. The great depression of the 1930s had about reached its bottom, nobody was interested in my surgical training because the doctors did not refer paying patients. I still have my first account book which shows that in the first six months of practice at no time did I have a gross income above $50 a month. Since I was already married and had 2 children this income obviously was not adequate.

In spite of the total lack of interest in my surgical training, I found that many of the doctors were asking me questions about physical therapy and it seemed that this field could be developed. Therefore, after nine months of general practice I decided to go into this new field as a specialty before I built up enough medical practice to worry about dropping it. Therefore, I rented an office in the Medical Arts Building and put my name on the door with the notation, "Practice limited to Physical Therapy."

Then I starved for three years.

After these years of living on borrowed money I began to be able to pay my office expenses and most of my living expenses so I hired a physical therapist to assist in my private work. Before that time I had been doing all of the therapy myself.

In 1935 Doctor Frank H. Krusen came to the Mayo Clinic to develop a department of physical therapy there. Then there were 2 specialists in physical therapy in the state of Minnesota.

In 1939 I was asked to take charge of the physical therapy department at the university. At that time the department was administratively under the department of radiology but was divided as mentioned before. The personnel consisted of Miss Kollman, the graduate physical therapist, Miss Laura Beeklund, the nurse assigned part-time from deep X-ray therapy, and Olga Olson, an aide.

Our first administration change was to unify the department under my direction.

Miss Kollman was very anxious to start a training course for physical therapists and we started preparations immediately to accomplish this. In 1940 our first class was enrolled. It consisted of 5 students, 3 men who were graduates in physical education and 2 women who were graduate nurses. The course was arranged to start with the summer session so that physiology, physics, anatomy, and other basic courses could be taken during the summer. The actual training in physical therapy techniques and patient care would begin with the fall quarter and end in the spring. We felt that we could handle a small class such as these 5 students. To our dismay when the registration for the summer session was completed there were more than 20 students registered. However, when the actual physical therapy began in the fall our fears were allayed because the original 5 students were the only ones who passed the courses. The rest were looking for easy courses. Our original students not only graduated but passed the national examination of the American Registry of Physical Therapists with flying colors.

There was no university budget for this course. Miss Kollman and Miss Becklund were salaried employees of the university but they had not only teaching duties but regular treatment duties to carry on. I was part-time and nonsalaried. Since there was no budget, I purchased any needed supplies with my own very limited funds. Applicants for the course increased and in 1944 Miss Ruby Green was added to the staff as technical director of the physical therapy course. She was a graduate of our class of 1943 and in order to qualify her as a teacher we sent her to Stanford University for a year where she received her Masters Degree.

Fever therapy developed about this time and in 1944 we were able to add a room in which a Kettering Hypertherm was placed and Doctor Malcolm Cook, who I believe had been a resident in pediatrics, was placed in charge of the

fever therapy. This program continued until 1952 when the use of penicillin and other antibiotics which were much safer, became more available and made fever therapy obsolete.

Candidates for the physical therapy course increased in number quite rapidly. From the first class which graduated in 1942, consisting of 5 members, we proceeded to 8 in the 1943 class. Then because of the pressure of the war, we stepped up our program so that we had 2 classes a year. By 1946 we had 26 students in the class.

In the meantime we had decided to start a degree course and gradually phase out the one-year certificate course. This required a great deal of negotiation with the College of Science, Literature, and the Arts, which insisted that we should impart some culture to our students as well as knowledge. Yet, our curriculum was so crowded with subjects required to prepare the students adequately for the practice of physical therapy that it was most difficult to try to find space for these cultural activities. Finally we made a compromise and included a course in humanities which seemed to satisfy the cultural requirements.

Doctor Catherine West, who had been my associate, resigned in 1945 to become a full-time mother and Doctor Alice Brill then took her place.

We had many problems including those of obtaining enough classroom space for teaching purposes not only for classroom teaching but also for clinical teaching.

The only available classroom was a small blood room across from the clinic which was miserably hot, small, and smelly. It contained a big refrigerator which made more noise than a threshing machine. This room was rather appropriately dubbed "The Dungeon" by the students. However, with their usual lighthearted enthusiasm they took even this in stride and came up with an ultraviolet theme song which started: "Alopecia ary-areata, acne vulgaris."

Affiliation with the Minneapolis General Hospital provided greater clinical facilities and a massage and corrective exercise laboratory.

The first degree course was graduated in 1948. These students obtained a Bachelor of Science degree as well as a certificate. We used the examination of the American Registry of Physical Therapists for our final and a student had to pass the registry examination in order to pass the course.

During the war also, a need was recognized for the establishment of an occupational therapy course and we had many meetings with various members of the university staff and representatives of the war effort in order to get this started. In 1946 Miss Borghild Hansen* came to the university to organize the course in occupational therapy which has expanded and prospered, and remains under her direction at this time.

During the war also, Doctor Frederic J. Kottke, who already had a Ph.D. in physiology as well as an M.D., was induced to take a Baruch fellowship in physical medicine, receiving training in a number of centers throughout the country. He then came back to the University of Minnesota in 1947 to become the full-time director of our department. Under his leadership it has expanded with remarkable speed and to undreamed of dimensions in all phases of teaching, research, and clinical practice.

To return to a little earlier level, in 1940 Sister Kenny came to the university and the Minneapolis General Hospital and the Kenny treatment was instituted, investigated, and expanded. An immediate demand for training in this revolutionary new technique was evident so courses were started with the financial assistance of the National Foundation for Infantile Paralysis, Inc. to train nurses, therapists, and doctors in the Kenny concept and treatment of poliomyelitis. This required a great deal of my time so that I was put on the payroll at more than 50% time. After Doctor Kottke's arrival he took over more and more of the work of the department so that my time and remuneration decreased year by year. At the same time my rank gradually rose so I had the interesting and somewhat frustrating experience of having my dues at the Campus Club increase according to my increasing rank but my income decrease according to my decreasing time involvement. The Club dues eventually took such a large part of my very limited salary and my time available for its use was so small that I resigned.

Soon after I assumed direction of the department at the University Hospital, attempts were made to introduce a required course in physical medicine into the medical school curriculum. At that time I believed that it was best to have this course during the senior year when the student would be more mature and probably more receptive to this type of informational material. However, I later found that this was not true, that the students during their senior year were rather blasé and uninterested and usually had already made up their minds as to what they felt was good or bad in medicine.

Merely finding a spot in the curriculum for these lectures was a major problem. Each department of the medical school felt that its time was

*Deceased Oct. 6, 1966

inadequate to present its own material and so each was looking for more time rather than less. After much negotiation and largely through the efforts and cooperation of Dean Diehl we were able to get one lecture a week for 1 quarter. However, at that time the senior lectures were given at 8 A.M. and each department had a day or two during the week assigned to it for these presentations. Therefore, in order to steal as little time from each department as possible, my lectures were staggered so that the first week the lecture was given on Monday, the next week on Tuesday, the next on Wednesday, and so forth through the week. After Saturday the lecture returned to Monday and the process was repeated. While this method raised havoc with my own schedule it reduced the antagonism of other departments to as low a level as possible.

Additional contact with the medical student has been introduced into the curriculum since then, so that at the present time there is some degree of contact in each year of the curriculum. Physical medicine staff members participate in teaching applied anatomy to the freshmen. Physical diagnosis as applied to physical medicine is taught to the sophomores. During the junior and senior years teaching is combined with care of clinic patients and in the senior year, the lectures are presented.

Probably one of the most effective methods of interesting medical students in physical medicine and rehabilitation has been the summer scholarships offered by various organizations, which have given remuneration to sophomore and junior students for study and experience in the department during summer vacation. This has served as a source of candidates for residencies and promoted interest in the field generally.

The residency program has grown considerably, although there is always a need for more residents and more specialists in the field. From 1 or 2 residents sometimes shared with other departments in the early days, the number of residents has increased to a maximum of 12 two years ago and, at present, 9 are receiving training.

Continued from page 132

TRANSFERS AND MOBILITY FOR PARAPLEGIC AND QUADRIPLEGIC PATIENTS

Roger P. Hallin, M.D.

in and out of a bathtub. These devices are particularly useful when patients are overweight or when for some other reason the helping individuals find it difficult to do the necessary lifting.

Once the transfer is accomplished, devices for getting about in the chair come into play. Vertical projections on the hand rims are helpful for many people who have no voluntary hand function. Some have difficulty with this arrangement, particularly on heavy rugs, in grass, and in inclement weather in snowy climates, and they may find that knobs attached on the hub side of the rim are more desirable. Paraplegics usually prefer plastic hand rims, now standard equipment on many models, which provide a certain amount of friction.

With automobile hand controls, a person who has learned to get in and out of bed, dress, get in and out of a wheelchair, and get into a car and pull his wheelchair in after him and drive away has achieved the mobility to become an independent citizen rather than an invalid. The quadriplegic, with much greater limitations, cannot reach the same level of independence but transfer methods can be evolved for him that require comparatively modest amounts of physical effort by an attendant.

The techniques described here are simple, but they are dependent for their usefulness upon attention to small details. Consideration of these problems and instruction in methods to overcome them can spell the difference between immobility and independence. This is a situation in which treatment leading to cure is not available, but restoration of functional activity of the patient is a most worthwhile goal for the physician's efforts. A disabled person who can get about either independently or with a minimum of help has broadened horizons which make it possible to live with purpose and to accept himself as a useful member of society.

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Frank H. Krusen, M.D.

Pathmarker in Physical Medicine and Rehabilitation

JAMES ECKMAN, Ph.D.

He it was that first gave to the law the air of a science. He found it a skelton, and clothed it with life, colour, and complexion. . . .

—LORD AVONMORE: *On Blackstone*

WHAT LORD AVONMORE said about Sir William Blackstone and the law might well be applied to Dr. Frank Hammond Krusen and physical medicine and rehabilitation. He found it a pale wraith indeed when, in 1929, with a staff of two therapists, he established the country's first department of physical medicine at Temple University.

Bit by bit he clothed the spare frame of the undeveloped specialty, setting up graduate seminars in physical medicine in Philadelphia, instituting a training program for medical students at Temple University, investigating the field as chairman of a committee on physical medicine of the Pennsylvania State Medical Society, and contributing papers on physical medicine to medical journals as well as a chapter on electrosurgery to Piersol's *Cyclopedia of Medicine* of 1934. He had always been keenly interested in the dissemination of knowledge about medicine. In his class at Temple, he made film strips depicting salient aspects of physical medicine; these he used as teaching tools. But a work which was a salient contribution to the entire domain of the latent specialty was his 186-page *Light Therapy*, published in 1933.

By 1935, the shadow cast by the once-infirm specialty of physical medicine was considerably larger than a man's hand. It had reached westward as far as the Mayo Clinic, which in May of that year invited Dr. Krusen to inaugurate a section devoted to the field. He accepted and, with the corollary appointment as associate professor of physical medicine in the Mayo Foundation, he began to apply himself, on the one hand, to graduate training in physical medicine in the form of fellowships in the Mayo Foundation and, on the other hand, to the practical training of physical therapists. The first fellowship in the specialty in the Mayo Foundation was established in 1936; the Mayo Clinic School of Physical Therapy was founded in 1938. A number of physiatrists, who today head departments of physical medicine and rehabilitation in medical schools, received their graduate training under Dr. Krusen. Graduates of the school of physical therapy are eligible for certification as registered physical therapists by the American Registry of Physical Therapists.

His diligence in contributions to the literature continue unabated. A small book, *Physical Therapy in Arthritis*, was published in 1937. In 1941, his *Physical Medicine*, which became the standard text in the field, was issued as a massive work of 846 pages and 351 illustrations. Dr. Krusen was editor of the *Yearbook of Physical Medicine and Rehabilitation* (1949 and 1950) and of *Physical Medicine and Rehabilitation for the Clinician* (1951). He is author or co-author of 7 volumes and more than 425 papers on various phases of his specialty. His most recent major editorial activity has been as senior editor

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of the *Handbook of Physical Medicine and Rehabilitation* (1965).

By 1942, the clinical practice of physical medicine at the Mayo Clinic had increased by 83% from the figure of 1935, and 84 students were in training in 5 separate courses. Personnel in the same period had increased from 18 to 108. As a prime figure in the specialty, Dr. Krusen was becoming known throughout the land. In 1943, he was invited to become executive director of the newly formed Baruch Committee on Physical Medicine. He held this post until 1951. A ten-year plan was evolved under which $1,100,000 was to be disbursed for the advancement of teaching and research in physical medicine in the United States. From this fund the University of Minnesota received $40,000 and, later, $15,000 was added to this grant. He also found time to stimulate research on the application of microwave diathermy and to investigate the possible application of ultrasonic energy to physical medicine.

In 1960, he was granted leave of absence from the Mayo Clinic to become president of the Sister Elizabeth Kenny Foundation and medical director of the Kenny Rehabilitation Institute. In less than three years, he was able to bring respect and dignity back to the foundation after a demoralizing period in which the good name and fortunes of the organization had been grievously wounded. When he retired as president on July 1, 1963, the foundation had regained public esteem. His retirement from the foundation coincided with his retirement from the Mayo Clinic, where he had been a senior consultant since 1958.

Not long afterward, he returned to Temple University as professor and coordinator of physical medicine and rehabilitation in the Temple University Hospital.

Throughout his professional career, Dr. Krusen has provided national and international leadership in his specialty. He was elected president of the American Academy of Physical Medicine and Rehabilitation when it was organized in 1936 for the purpose of developing physical medicine into a recognized medical specialty. As might well be expected, he was one of the founders of the American Board of Physical Medicine and Rehabilitation and the first chairman, serving from 1947 to 1949. He has been the treasurer of the American Congress of Physical Medicine and Rehabilitation since 1950. He has served as chairman of the Council on Medical Physics and of the Committee on Rehabilitation of the American Medical Association (AMA). Dr. Krusen was chairman of the Committee on War and Post-War Rehabilitation. In 1959 and 1960, he served as special assistant for health and medical affairs to the director of the Office of Vocational Rehabilitation of the Department of Health, Education, and Welfare. He has been a member of the Committee on Rehabilitation of the American Heart Association and a counselor to the National Society for Crippled Children and Adults. He is the senior consultant on physical medicine to the surgeon general of the United States Army.

In 1950, Dr. Krusen was a founder and the first president of the International Federation of Physical Medicine. In 1960, he was the president of the Third International Congress of Physical Medicine held in Washington, D. C., and, following that, served another four-year term as president of the International Federation of Physical Medicine.

In Minnesota, Dr. Krusen was president of the State Board of Health from 1955 until his retirement in 1963. He has been chairman of the Governor's Advisory Committee on Vocational Rehabilitation. Since 1958, he has served as chairman of the Expert Medical Committee of the American Rehabilitation Foundation.

Virtually every honor which could come to a man in his field has been conferred upon Dr. Krusen, with an impressive number from outside his specialty. Of the latter, one of the most notable is the Distinguished Service Award of the AMA in the form of a gold medal, presented to him in 1958. Another is the Physician's Award for 1953, signed by the President of the United States, for outstanding contributions in promoting the rehabilitation and employment of the handicapped.

On October 19, 1966, the Frank H. Krusen Center for Physical Medicine and Rehabilitation was dedicated at Temple University. It is a 9,200–sq. ft. edifice built at a cost of $405,000 and designed for the rehabilitation of patients stricken with an impressive variety of disabling diseases.

Other awards from within his specialty and from medicine in general are the gold key of the American Congress of Physical Medicine and Rehabilitation (1944), the Pennsylvania Ambassador Award, and the *Modern Medicine* Award for Distinguished Achievement (1956). He was elected vice-president of the staff of the Mayo Clinic in 1957, and a year later he was made a fellow of the American Medical Writers' Association. In 1958, he received a citation from the governor of Minnesota for meritorious services to the state and nation. In 1960, he was elected to honorary membership in the French

National Society of Physical Medicine. In the same year, the Connecticut Society of Physical Medicine presented to him a gold medal bearing his likeness and inscribed to him as "pioneer in research, acclaimed as a teacher, clinician and statesman, but best remembered as an inspiration to young physicians."

In 1961, Dr. Krusen was cited by the Goodwill Industries of America for his outstanding services to the handicapped, and the following year, he was among those honored at the Salute to Excellence ceremonies of the Academy of Achievement at San Diego. On July 2, 1962, he received the honorary degree of doctor of medicine from the Justus Leibig University at Giessen, Germany. In August of the same year he was made an honorary member of the University of Chile Faculty of Medical Sciences, and the department of physical medicine and rehabilitation at the L. Calvo McKenna Hospital in Santiago de Chile was given his name as an honor to him. In 1963, he served as honorary president of the fiftieth annual meeting of the Belgian Society of Physical Medicine at Brussels.

Dr. Krusen is a fellow of the American College of Physicians, and a member of the World Medical Association, American Association for the Advancement of Science, AMA, American Rheumatism Association, American Academy and Congress of Physical Medicine and Rehabilitation, American Association of University Professors, and Sigma Xi. He is an honorary fellow of the Royal Society of Medicine of England, the British Association of Physical Medicine, the Danish Society of Physical Medicine, the Argentine League Against Rheumatism, the Argentine Society of Physical Medicine, the Chilean Society of Physical Medicine and Rehabilitation, the Chilean Society of Rheumatology, the Chilean Society of Rehabilitation, and the Howitt Club of London. In addition, he is an honorary member of the societies of physical medicine and rehabilitation of Austria, Belgium, Brazil, Canada, and Peru. He was president of the Mayo Foundation chapter of Sigma Xi in 1958 and 1959.

Dr. Krusen was married to Margaret Louise Borland on May 28, 1921. Dr. and Mrs. Krusen have two daughters: Joanne Elizabeth (Mrs. Robert M. Hart of New Canaan, Conn.) and Janice Alison Krusen of Philadelphia.

In lighter moments of rare leisure, Dr. Krusen has found some time for the sock and buskin. He had a part in *Antigone*, as presented by the Rochester Civic Theater, and he directed *My Three Angels* for the same group. He played the part of a fisherman in *The Witchfinders*, and in *The Moon Is Blue* he was cast as the father of his daughter, Janice, who had the leading role in the play.

Coming in June

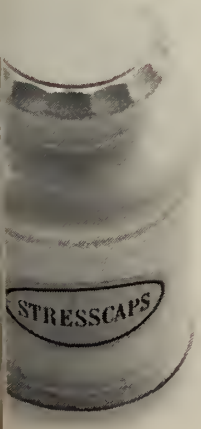
Symposium — Physical Medicine and Rehabilitation — Part II

- Physiology of Balance *by Frederic J. Kottke, M.D., Minneapolis*
- Perceptual Problems of the Hemiplegic Patient *by Robert R. Sawtell, M.D., and Gordon M. Martin, M.D., Rochester, Minn.*
- Problems of the Bladder and Bowel in Patients with Myelopathies *by Ann H. Schutt, M.D., Thomas L. Pool, M.D., and Gordon M. Martin, M.D., Rochester, Minn.*
- The Relationship of the Staff Physician to the Department of Physical Medicine and Rehabilitation of a Community Hospital *by Miland E. Knapp, M.D., Minneapolis*
- History of Physical Medicine at the University of Minnesota from 1945 to 1965 *by Mildred Olson, B.S., M.T. (A.S.C.P.), Minneapolis*



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Vitamin B_{12} Crystalline	4 mcgm
Vitamin C (Ascorbic Acid)	300 mg
Niacinamide	100 mg
Calcium Pantothenate	20 mg

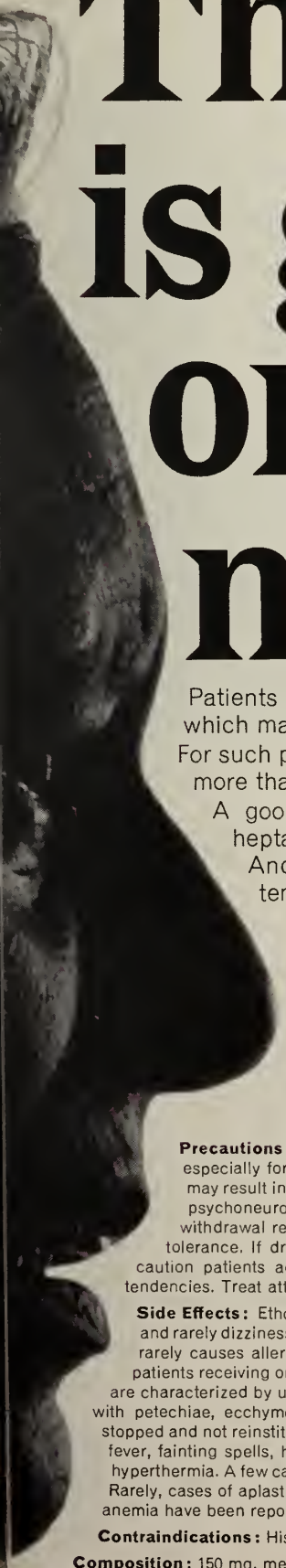
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Book Reviews . . .

Antibiotics and Chemotherapeutic Agents in Clinical and Laboratory Practice

by VICTOR LORIAN, M.D., *337 pp., ill., Charles C Thomas, Springfield, 1966. $15.50*

Although this book is designed to be a practical guide for the use of chemotherapeutic drugs in infections by physicians and laboratory workers, it succeeds only in part.

The book begins with a discussion of some of the factors which might determine the choice of an antibiotic for a particular type of infection. Next, there is a brief and inadequate review of bacterial drug resistance. Recommendations are then given for therapeutic regimens preferred by the author in the more common types of microbial infections. His choices are generally sound but his directions for administration are often confusing in regard to dosage, and the duration of therapy is often neglected. In some instances, he prefers antibiotics which are not available in the U.S. Also, some readers might quarrel with the emphasis placed upon endobronchial treatment for pulmonary infections.

There is a useful and interesting discussion of some practical aspects of laboratory tests for bacterial resistance to antibiotics. The presentation of data on disk-diffusion methods will be especially helpful because a lack of understanding of the technical variable is widespread.

More than half the pages are devoted to a review of the practical therapeutic aspects of the available antibiotics and chemotherapeutic drugs. Physical properties, bacterial susceptibility, pharmacology, indications, and side effects are given succinctly. In the case of some of the new penicillinase-resistant penicillins, cephalothin, gentamycin, and other rather new antibiotics, the reviews are too sketchy to be worthwhile.

The book has an adequate index. Unfortunately, the writing is often awkward and words are frequently misspelled. The most recent references in the bibliography are in the year 1965. This book will be most useful in diagnostic bacteriology laboratories. Physicians concerned with treatment of infections may find it a good source of practical information about antibiotics, particularly those which have not come into use in the past two years.

WENDELL H. HALL, M.D.
Minneapolis

Atlas of Hernia Repair

by CARL H. CALMAN, M.D., *159 pp., ill., C. V. Mosby Co., St. Louis, 1966. $16.75*

This atlas fills a small but important niche in the training of an embryo surgeon and provides a review for his instructor. As Dr. Calman states it has been customary to regard surgery of hernias as relatively simple and thus the resultant instruction has not been always as adequate as it should be. In addition, the end results speak forcefully for this statement and thus serve as a stepping-stone for the launching of this text.

It is evident that the author has spent considerable time in abstracting the literature as well as recent excellent surgical texts on this subject. Thus, the author



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has presented the reader with an admirable abstract of current surgical thought. The author proceeds from the discussion briefly of the anatomy and types of hernias through the chapters on direct and indirect inguinal hernias to the extremely rare pelvic floor and internal hernias. The subject of the direct and indirect inguinal hernia is well discussed, including the complications of the procedure that may follow. All the major repair methods, such as the Halsted, Bassini, and Andrews as well as the McVay, are well depicted and described in the accompanying text. The modifications of hernia repairs in children are also thoroughly discussed. The chapter on the anatomy and repair of sliding hernias constitutes a well-depicted inclusion in this book. The method adds considerably to surgical confidence when such a hernia is encountered. The illustrations in all chapters are of very good quality, although in a few instances one would wish them to be enlarged for ease of review.

The author is frank to admit when he is not familiar with a procedure even though he will discuss it for the sake of completeness. The quality of the paper is excellent, and the book evidently has been carefully edited before publication.

This reviewer feels that the chapter on esophageal hiatus hernias was too simplified; however, it would serve as a very good review for those who are familiar with the problems encountered. The terse description of the procedures in other chapters, however, serves admirably for rapid review.

The appearance of the book is attractive and its contents can be easily reviewed in several evenings. I feel that it is an excellent purchase for the senior medical student, intern, and resident surgeon and should prove worthwhile to the practicing surgeon.

E. F. BOLLIGER, M.D.
Ellendale, N.D.

Radiographic Atlas of the Genitourinary System

by CHARLES NEY, M.D., *and* RICHARD M. FRIEDENBERG, M.D., *741 pp., ill., J. B. Lippincott Co., Philadelphia, 1966. $36*

The book is an ambitious effort, on the part of a urologist and a radiologist, aided by ten contributing authors, to assemble an encyclopedic collection of radiographs illustrating almost every known normal variation and pathological entity affecting the urinary and genital systems.

The authors "stress pictorial roentgenographic findings" and "limit the text as much as possible." This, I think, is unfortunate since it limits the usefulness of the work as a source of information for those who want and need it most—the urologist or radiologist in training. What the book lacks in text is partially compensated by the succinct captions. The bibliography is comprehensive, but recent pertinent references are few in number.

The quality of reproductions of the radiographs is, in most instances, satisfactory or good; in others clarity of detail leaves something to be desired—particularly when compared with those found in urological and radiological journals.

One is bound to compare this work with others similar in content. Emmett's *Clinical Urography* (Saunders, 1964), which is billed as an "atlas and textbook," has considerably more text, including valuable sections de-

(Continued on page 26A)

(Continued from page 25A)

scribing numerous techniques for delineation and evaluation of various portions of the urinary and genital tracts.

This volume should be a useful addition to the appropriate departmental libraries of universities and hospitals and should be available to trainees in urology and radiology as well as practicing urologists, radiologists, and selected generalists.

FRANK D. NAEGELI, M.D.
Minot, N.D.

Monograph of the Pasteur Institute: Manual for the Classification and Determination of the Anaerobic Bacteria

by ANDRE R. PREVOT, *1st American edition, (translated from the third French edition) 402 pp., Lea & Febiger, Philadelphia, 1966. $15*

Difficulties encountered by diagnostic bacteriologists in the identification of anaerobes is undoubtedly due to a lack of familiarity with methodolgy. The author stresses the importance of using anaerobic as well as aerobic methods in routine culture techniques. Using habitat, morphology, physiology, cultural characteristics, biochemical properties, and pathogenicity as criteria, Prévot presents a clear and concise scheme for the identification of this group of organisms. The first section clearly outlines the method of classification used. Keys for the identification of the anaerobes are grouped in the remaining section. This appears to be much less cumbersome than the method of identification encountered in Bergey's Manual, where the anaerobes and aerobes appear together.

The introduction of Prévot's system of classification will cause concern among students of bacterial taxonomy who have been trained according to the Bergey system. Prévot considers the bacterial kingdom as an autonomous branch of the biological world, a concept based on structures and chemical components unique to the bacterial cell. In contrast, Bergey's classification places the bacteria in the plant kingdom. Although major taxonomic differences exist between the Prévot and Bergey systems, the genus and species designations are similar. Where minor discrepancies exist, final identification according to the Bergey system is listed to avoid confusion. The classification of Clostridium perfringens will serve as an example of the differences in taxonomy between these systems.

Bergey System		Prévot System	
Kingdom	Plant	Branch	Schizomycetes
Division	Protophyta	Subbranch	Eubacteria
Class	Schizomycetes	Class	Sporulales
Order	Eubacteriales	Order	Clostridiales
Family	Bacillaceae	Family	Clostridiaceae
Genera	Clostridium	Genera	Welchia
Species	perfringens	Species	perfringens

Unless one is a student of pure bacterial taxonomy, it is difficult to assess the merits of each system of classifi-

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cation. Though each system incorporates changes in classification advocated by the other, with due credit given, it is apparent that a universal system must be adopted.

This book should be included in the references used by the hospital diagnostic bacteriologist. In spite of the differences advocated by Prévot in bacterial taxonomy, per se, this manual will serve as a valuable tool for the identification of anaerobic bacteria.

RICHARD H. LYON, PH.D.
Minneapolis

Carotid-Cavernous Fistula

by WALLACE B. HAMBY, M.D., *139 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $7.50*

This is an excellent book covering a small but important aspect of vascular problems. It is extremely well written in clear concise language and has been edited so that it can be read rapidly without sacrificing important material. The book is well illustrated with photographs, drawings, and diagrams. It should be recommended for students, residents, and those interested in vascular problems of the head and neck.

W. H. REMINE, M.D.
Rochester, Minn.

Comparative Leukaemia Research: Proceedings of an International Wenner-Gren Symposium held in Stockholm, September 1965

edited by G. WINQVIST, *268 pp., ill., Pergamon Press, New York City, 1966. $13.50*

This volume is a compilation of the proceedings of an international symposium on the cause and nature of leukemia in man and animals held at the Wenner-Gren Center in Stockholm. The symposium was designed to present research in three areas: virology, immunology, and epidemiology.

The sections on viruses and leukemia includes a basic discussion of known interactions between tumor viruses and cells, a series of papers on factors which appear to influence viral leukemogenesis, and a number of specific studies on bovine, murine, and avian leukemias.

The section on tumor immunology is introduced by a general summary of the problem of tumor-specific antigenicity and how this property might be used for immunotherapy or prophylaxis. These considerations are further elaborated in discussion of the prevention and treatment of virus-induced cancers in experimental animals. It is pointed out that so far no procedures are available which have application to malignant diseases in man.

A possible role of autoimmune mechanisms in leukemogenesis is considered in a relatively brief fashion. This includes a review of some of the general concepts of autoimmunity and speculation as to the possible role of such mechanisms in malignant disease.

Considerable discussion is devoted to epidemiologic aspects of human and mammalian leukemia in relatively detailed reviews, plus a series of shorter discussions of the epizootiology of leukoses in birds, wild animals, cattle, dogs, and so on.

(Continued on page 31A)

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News Briefs . . .

North Dakota

DR. PETER VERNON MORRISON, originally of London, England, is practicing medicine in Glen Ullin.

* * * *

DR. GORDON MAGILL was elected chief of staff at St. John's Hospital in Fargo.

* * * *

DR. HAROLD TARPLEY of Grand Forks was named president of the Third District Medical Society.

* * * *

DR. LEONARD W. LARSON of Bismarck has been named a special medical representative of the American Cancer Society to promote annual checkups for cancer detection.

* * * *

DR. C. H. PETERS has been elected chairman of the board of directors at Quain and Ramstad Clinic in Bismarck.

* * * *

DRS. C. J. GLASPEL, W. P. TEEVENS, and L. P. EATON of Grafton have moved to the new Grafton Clinic building.

* * * *

DR. BERNARD B. HUSS, JR., of Minot has been elected to active membership in the American Academy of General Practice.

* * * *

DR. N. J. KALUZNIAK of Langdon has been named vice-president of the Third District Medical Society.

* * * *

DR. WILLIAM C. NELSON was elected chief of staff and president of the executive board of St. Michael's Hospital in Grand Forks.

Minnesota

DR. THEODORE LARSON has joined the staff of the Northern Pines Mental Health Center in Little Falls as psychiatrist.

* * * *

DR. DONN G. MOSSER, a Minneapolis radiologist, has been appointed campaign leader for the 1967 Cancer Crusade in Minnesota.

* * * *

DR. JOHN M. SCANLAN has joined the staff of the Fergus Falls State Hospital.

* * * *

DR. GEORGE MIKS has terminated his practice in Eveleth.

* * * *

DR. RALPH G. HIRSCHOWITZ will furnish psychiatric services to the comprehensive community center at Northwestern Hospital in Thief River Falls.

* * * *

DR. JOHN H. AGA was named chief of the medical staff at St. Joseph's Hospital in Brainerd.

* * * *

DR. MATHEW J. WEIR, Virginia, has been honored by the American Medical Association for his volunteer work in Vietnam.

* * * *

DR. CHARLES W. ROTH, an internist, has joined the staff at Medical Block Clinic in Red Wing after twenty-two years of active military service.

* * * *

DR. ARNOLD ANDERSON, a St. Louis Park pediatrician, has been appointed director of Children's Hospital of Minneapolis.

DR. KARL E. JOHNSON has been named chief of staff of St. Luke's Hospital in Duluth.

* * * *

DR. CLAYTON BENNETT, Rochester, was elected president of the Southeast Chapter of the Academy of General Practice.

* * * *

DR. MILO L. HANSEN has been elected president of the medical staff of St. Gabriel's Hospital in Little Falls.

* * * *

DR. WILLIAM E. JACOTT has been appointed to the University of Minnesota, Duluth branch, health service staff.

* * * *

DR. RICHARD H. SEIFERT has been elected chief of the medical staff at St. Ansgar Hospital in Moorhead.

* * * *

DR. I. T. CLARK has been named chief of the medical staff of St. Mary's Hospital in Duluth.

South Dakota

DR. WILLIAM BORMES, Aberdeen, was elected president of the board of directors of the Northeastern South Dakota Mental Health Center.

* * * *

DR. J. W. DONAHOE has been elected chief of staff at McKennan Hospital in Sioux Falls.

Deaths . . .

DR. JESSE P. NEHRING, 63, Preston, Minn., died Feb. 1. Dr. Nehring was Fillmore County coroner for over twenty-four years.

* * * *

DR. ERNEST A. OLSON, 66, Pine Island physician for the past forty-three years, died Jan. 23. Dr. Olson was a graduate of the University of Minnesota.

* * * *

DR. VICTOR M. VAUGHAN, 68, Truman, Minn., died Feb. 26. Dr. Vaughan was a graduate of the University of Minnesota.

* * * *

DR. JOHN P. FREEMAN, 90, died Feb. 2. Dr. Freeman was a graduate of the University of Illinois and practiced in Glenville, Minn., until his retirement in 1950.

* * * *

DR. JOHN A. WINTER, 87, Duluth, Minn., died Feb. 15. Dr. Winter was a graduate of Johns Hopkins University and was one of the founders of the Duluth Clinic.

* * * *

DR. RAYMOND T. HEALEY, 87, a Pierz, Minn., general practitioner, died Feb. 14. Dr. Healey was a graduate of the University of Minnesota and had been active in his practice until six months before his death.

* * * *

DR. ARNOLD LARSON, 73, a Detroit Lakes, Minn., general practitioner died Jan. 25.

* * * *

DR. ROBERT W. HOLMEN, 60, St. Paul otolaryngologist and ophthalmologist, died Feb. 10. Dr. Holmen graduated from the University of Minnesota and was on the boards of managers of Bethesda Hospital and Invalid Home.

* * * *

DR. WILLIAM H. FRITZ, 59, of Mitchell, S.D., died Jan. 26. Dr. Fritz, an ophthalmologist, was a graduate of Creighton University.



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(Continued from page 27A)

This volume represents a good compendium of current thought in these three areas of leukemia research. Although some of the material is of a rather general nature, it will be of interest mainly to those knowledgeable and experienced in these fields of research. It will be of less interest to clinical investigators and practicing clinicians.

ROBERT A. GREEN, M.D.
Minneapolis

One World of Science: Personal Visits to Men of Research in Many Lands

by WARREN ANDREW, M.D., *271 pp., ill., Charles C Thomas, Springfield, 1966. $8.75*

During the twenty-two years from 1940 to 1962, Dr. Warren Andrew, chairman of the anatomy department of the University of Indiana medical school, traveled and kept a journal in which he recorded impressions and details of visits with distinguished medical researchers the world over. More recently, he has expanded his journal notes and published them in this collection of reminiscences.

Dr. Warren writes about experiences in Prof. Oskar Vogt's German laboratory and about his host, who dissected Lenin's brain; conversations in London with Sir Alexander Fleming, discoverer of penicillin; visiting with Dr. Giuseppe Levi, Italy's leading microscopist; touring the laboratory of Dr. Tsunetaro Fujita, Japan's leading anatomy professor; and exchanging ideas with Dr. Mohammed Ishaq, Pakistan anatomist. He tells of similar experiences in such countries as Italy, Switzerland, Holland, Thailand, and India. He describes Dr. Edmund V. Cowdry of St. Louis as a "statesman of science" and refers to Dr. Edmond J. Farris of Philadelphia as "the stork caller" because of his research on sterility.

Dr. Warren includes vignettes of scientists who further the profession, delineating their research—and he does more. He manages with skill to convey the sights, sounds, and smells of countries and cities. The ways of people delight him. The result is an appealing book marked not only by the objective skill of a trained observer but also characterized by the humanist's subjective insight. An unusual travel book, it instructs and entertains in fascinating style.

PHILIP D. JORDAN, PH.D.
Minneapolis

Medical Care of the Adolescent

by J. ROSWELL GALLAGHER, M.D. *(with 8 contributing authors) 2nd ed., 489 pp., ill. Appleton-Century-Crofts, New York, 1966. $12*

The second edition of this unique book covers in a well-organized, comprehensive way the physical and emotional problems and peculiarities of the adolescent. Existing material has been thoroughly revised and guest authors contribute several new chapters in this edition. However, the major portion of the book is still written by the chief author who heads the adolescent unit of

(Continued on page 32A)

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FREE PARKING

(Continued from page 31A)

Childrens' Hospital Medical Center, Boston, and is clinical professor of pediatrics at Harvard's medical school.

Care of the adolescent has long been a no-man's-land, where the child has outgrown the pediatrician and has not become acquainted with an internist or where the busy general practitioner finds it difficult to give sufficient emphasis to this age group. Gallagher's clinic for adolescents in Boston has done much to clarify the medical and emotional needs of the teen-ager and to initiate a subspecialty in adolescent medicine, under the aegis of pediatrics for the most part.

His book should be read from beginning to end in order not to miss valuable ideas and suggestions. For example, the chapter on the office visit describes the best type of office arrangement and procedure, stressing that the emphasis be placed on the patient as a person and not just as a disease to be treated. Physical examinations, physical fitness, and sexual and physical development are covered in detail, including such conditions as acne, menstrual disorders, obesity, athletic injuries, orthopedic and postural defects, and epilepsy.

In my opinion, the reading of *Medical Care of the Adolescent* should have high priority for any physician treating adolescent patients.

LAURENCE G. PRAY, M.D.
Fargo, N.D.

NEW BOOKS RECEIVED

One World of Science: Personal Visit to Men of Research in Many Lands
by WARREN ANDREW, M.D., 271 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $8.75

Management of Amenorrhea
by CHARLES J. COLLINS, M.D., 308 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $11.75

The Artful Practice of Medicine
edited by WILLIAM B. D. VAN AUKEN, M.D., 115 pp., Charles C Thomas, Springfield, Ill., 1966. $6.50

Obstetrics and Gynecology
by J. ROBERT WILLSON, M.D., CLAYTON T. BEECHAM, M.D., ELSIE REID CARRINGTON, M.D., 3rd ed., 776 pp., ill., C. V. Mosby Co., St. Louis, 1966. $15.50

Explorations in Typing Psychotics
edited by MAURICE LORR, 241 pp., Pergamon Press, New York City, 1966. $9.25

Variation in Chemical Composition of the Nervous System as Determined by Developmental and Genetic Factors
edited by G. B. ANSELL, 119 pp., Pergamon Press, New York City, 1966. $4

Persistent Persecutory States of the Elderly
edited by FELIX POST, M.D., 100 pp., Pergamon Press, New York City, 1966. $4

Appraisal of Current Concepts in Anesthesiology
edited by JOHN ADRIANI, M.D., vol. 3, 522 pp., C. V. Mosby Co., St. Louis, 1966. $10.85

Food and Civilization: A Symposium
edited by SEYMOUR M. FARBER, M.D., NANCY L. WILSON, and ROGER H. L. WILSON, M.D., 341 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $11.75

Bone Tumors: General Aspects and Data on 3,987 Cases
by DAVID C. DAHLIN, M.D., 2d ed., 285 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $16

BOOK REVIEWS

Ocular Therapy: Complications and Management
edited by IRVING H. LEOPOLD, M.D., 157 pp., ill., C. V. Mosby Co., St. Louis, 1966. $11

Atlas of Cryosurgical Techniques in Ophthalmology
by CHARLES D. KELMAN, M.D., 118 pp., ill., C. V. Mosby Co., St. Louis, 1966. $18.75

Histones: Their Role in the Transfer of Genetic Information: Ciba Foundation Study Group No. 24
edited by A. V. S. DE REUCK and JULIE KNIGHT, 115 pp., ill., Little, Brown & Co., Boston, 1966. $3.50

Egg Implantation: Ciba Foundation Study Group No. 23
edited by G. E. W. WOLSTENHOLME and MAEVE O'CONNOR, 112 pp., ill., Little, Brown & Co., Boston, 1966. $3.50

The Thymus: Experimental and Clinical Studies: Ciba Foundation Symposium
edited by G. E. W. WOLSTENHOLME and RUTH PORTER, 538 pp., ill., Little, Brown & Co., Boston, 1966. $15

Touch, Heat and Pain: Ciba Foundation Symposium
edited by A. V. S. DE REUCK and JULIE KNIGHT, 389 pp., ill., Little, Brown & Co., Boston, 1966. $14

Chemotherapy of Disseminated Solid Tumors
by FRED J. ANSFIELD, M.D., 128 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $7.50

Practical Endocrinology
by JAMES H. HUTTON, M.D., 99 pp., Charles C Thomas, Springfield, Ill., 1966. $5.50

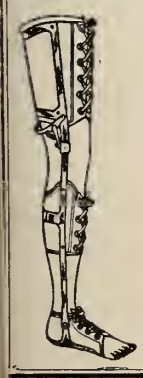
Physical Activity and the Heart: Proceedings of a Symposium, Helsinki, Finland
edited by MARTTI J. KARVONEN, M.D., and ALAN J. BARRY, Ph.D., 405 pp., Ill., Charles C Thomas, Springfield, Ill., 1966. $15.50

Unusual Clinical Features of Cirrhosis and Primary Liver Cell Carcinoma
by LUIS LEON-SOTOMAYOR, M.D., and VICTOR A. MOORE, M.D., 172 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $9.50

Reticuloendothelioses in Childhood: A Clinical Survey
by HAROLD W. K. DARGEON, M.D., 127 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $8.50

Anticancer Agents
by FRANCES E. KNOCK, M.D., 272 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $15.50

Prevention of Ischemic Heart Disease: Principles and Practice
edited by WILHELM RAAB, M.D., 466 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $25.50



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1 Sherman, L. F.: Minnesota Med. 48,605. May, 1965. 2 Sherman, L. F. Personal Communication.

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Description: CORTENEMA Hydrocortisone Retention Enema is a completely disposable, single-dose unit consisting of a flexible plastic container with a lubricated applicator tip for rectal administration. It is designed to permit self-administration and, therefore, it is suitable for both out-patient and hospital use. It also may be diluted with isotonic saline for administration by intrarectal drip.

CORTENEMA consists of a suspension of partially solubilized hydrocortisone alcohol in an aqueous vehicle. Thus, it provides the potent anti-inflammatory effect characteristic of the corticosteroids. Because hydrocortisone alcohol is absorbed to some extent from the colon, CORTENEMA provides both topical and systemic actions.

Indications: CORTENEMA Hydrocortisone Retention Enema is indicated as an adjunct in the treatment of idiopathic nonspecific ulcerative colitis. It is most useful in cases involving the rectum, sigmoid and left colon, where it exerts a topical effect. Due to its systemic action, CORTENEMA has proved effective in certain cases extending to the transverse and ascending colons.

Rectal steroid therapy brings about prompt symptomatic and proctologic remissions in a significant proportion of patients, but it apparently does not alter the recurrent and progressive nature of ulcerative colitis. Therefore, this form of treatment is not recommended for prophylaxis during periods of remission.

Contraindications: Local contraindications to the use of intrarectal steroids include obstruction, abscess, perforation, peritonitis, fresh intestinal anastomoses, extensive fistulas and sinus tracts. Tuberculosis (active, latent or questionably healed), ocular herpes simplex, and acute psychosis are usually considered absolute contraindications to the use of corticosteroids.

Relative contraindications include active peptic ulcer, acute glomerulonephritis, myasthenia gravis, osteoporosis, diverticulitis, thrombophlebitis, psychic disturbances, pregnancy, diabetes, hyperthyroidism, acute coronary disease, hypertension, limited cardiac reserve, and local or systemic infections, including fungal or exanthematous diseases. Where these conditions exist, the expected benefits from CORTENEMA must be weighed against the risks involved in its use.

If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

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Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

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Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of the extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.

SEARLE *Research in the Service of Medicine*

The Journal-Lancet Volume 87, No. 5, MAY 1967

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CONTENTS

Prefatory Remarks 143
Richard W. Olmsted, M.D.

Transfusion Therapy in Factor VIII Deficiency 143
George W. Dana, M.D.

Combined Immunosuppressive Therapy in Steroid-Resistant, Chronic Glomerular Diseases of Childhood 149
Robert A. Campbell, M.D., and Elsa Y. Jacinto, M.D.

The Physiologic Basis for the Supportive Management of the Sick Newborn 157
Richard E. Behrman, M.D.

Phenylketonuria: Report of the Oregon Detection and Evaluation Program 162
Janet M. Stewart, M.D., and Carl C. Ashley, M.D.

Classification and Treatment of Seizure Disorders in Children 167
Peggy J. Copple, M.D., and John B. Isom, M.D.

Determination of Gestational Age in Premature Infants 174
S. Gorham Babson, M.D., and C. M. McKinnon, B.S.

Heart Failure in Infancy 178
Martin H. Lees, M.D., and R. Clifton Way, M.D.

Electroencephalographic Responses to Sound and to Light in Premature and Full Term Neonates 181
Rudolf Engel, M.D.

Book Reviews 17A

News Briefs 20A

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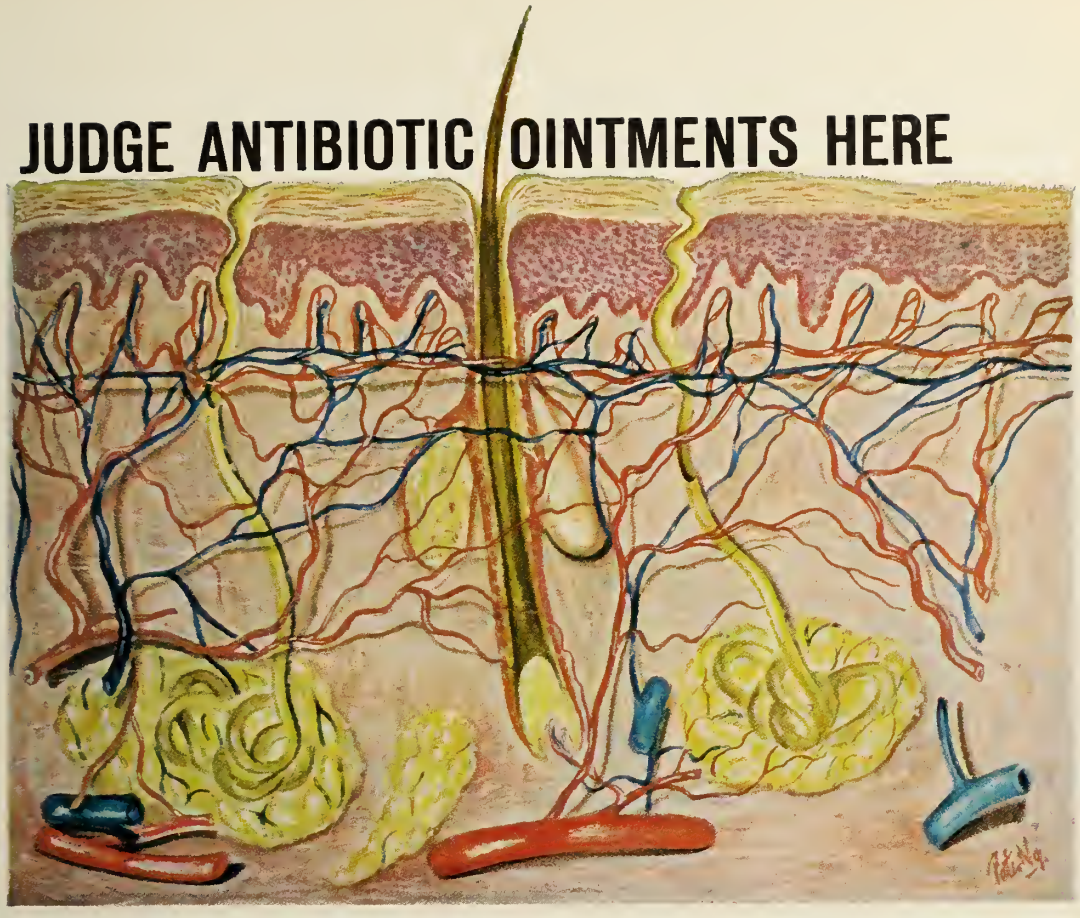
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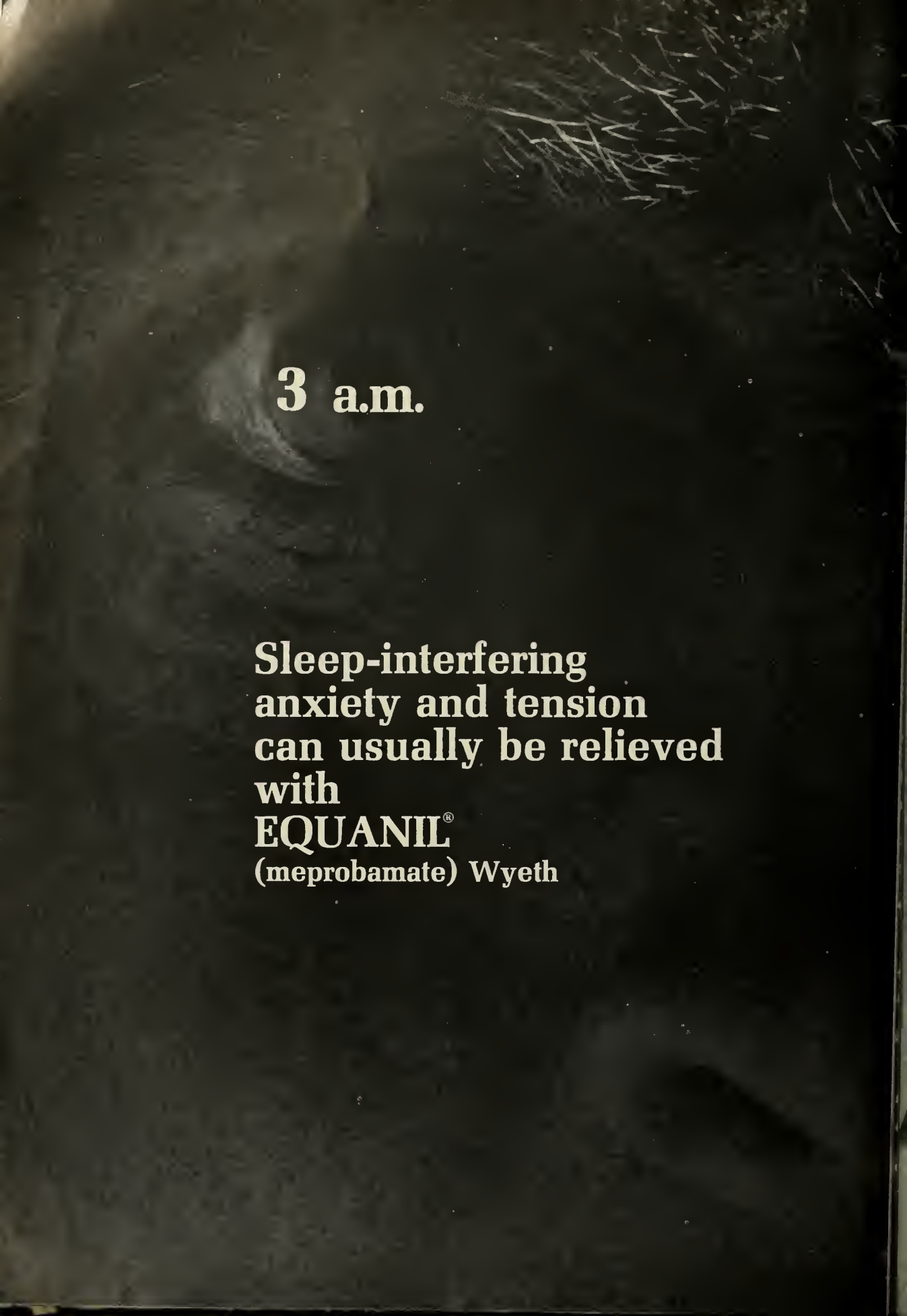
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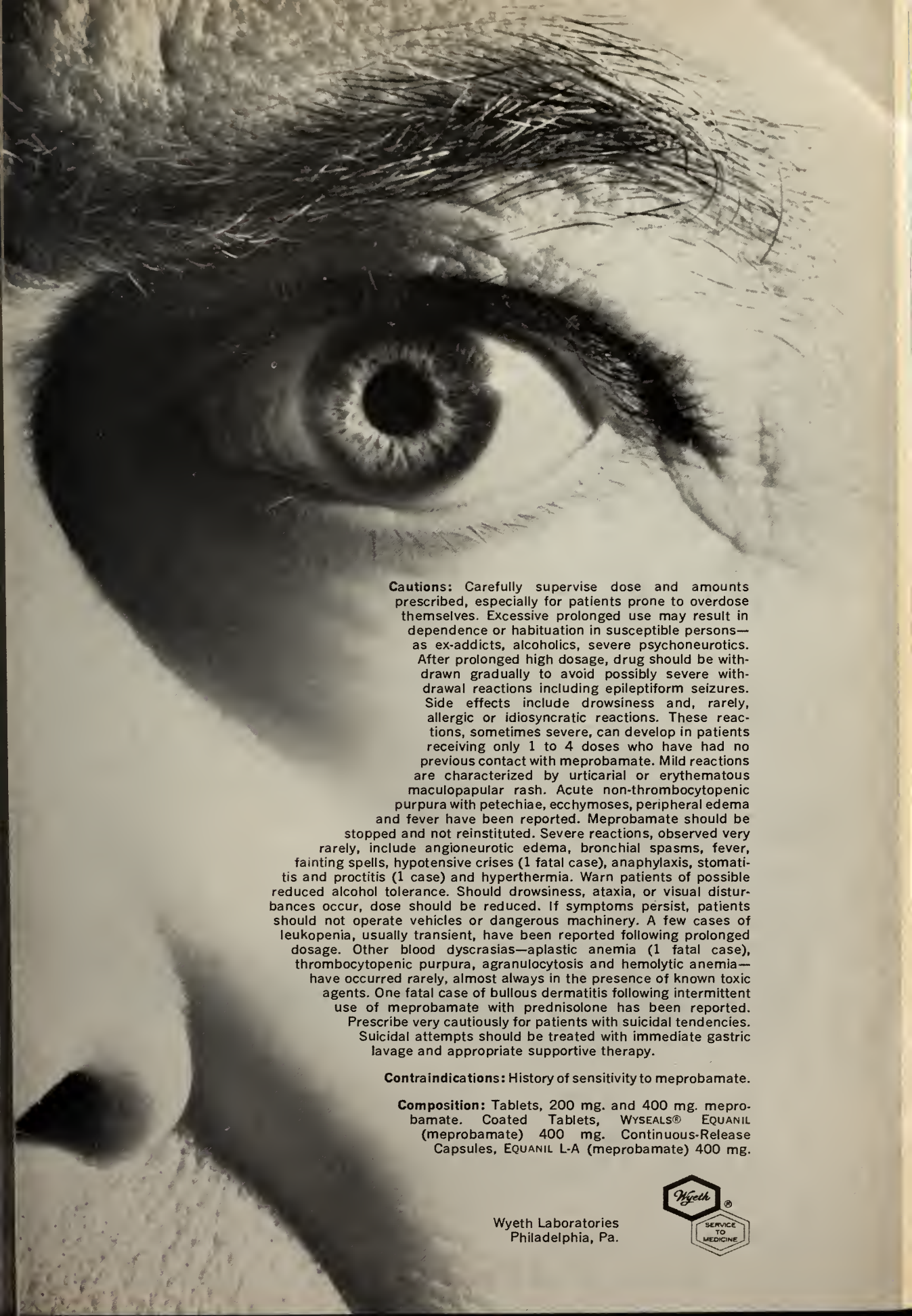
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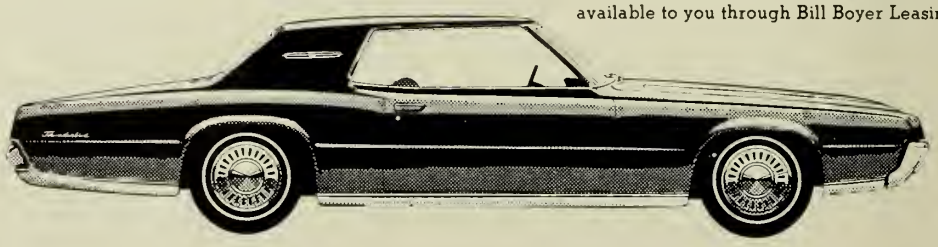
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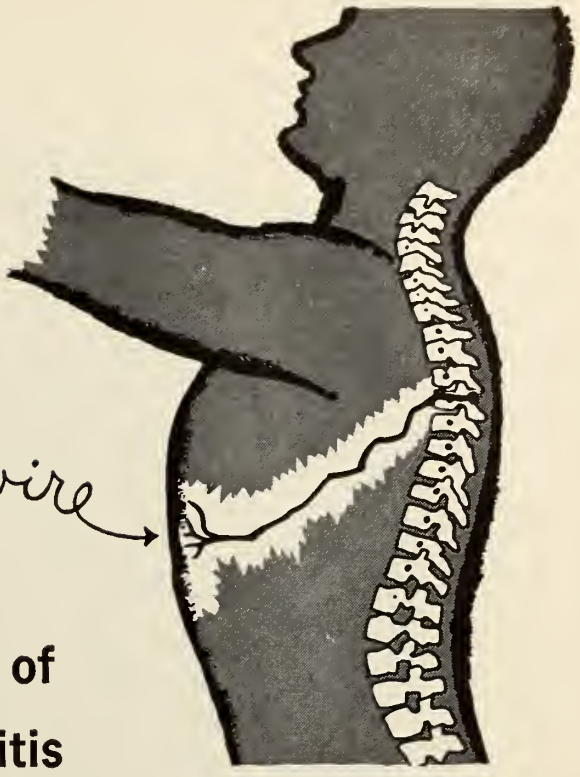
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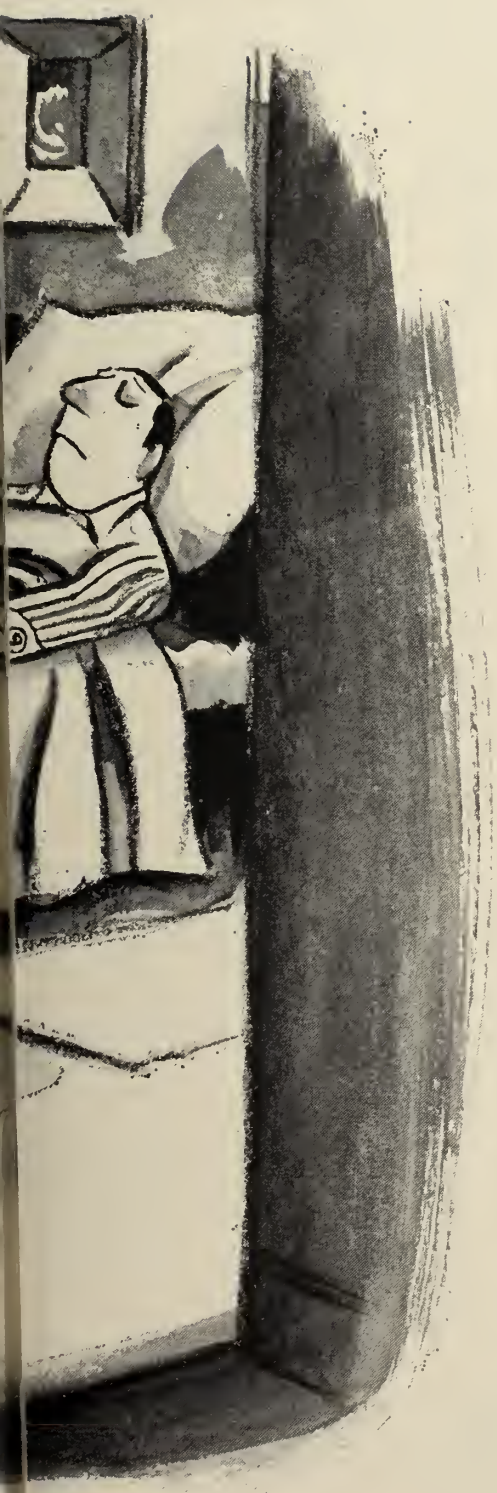
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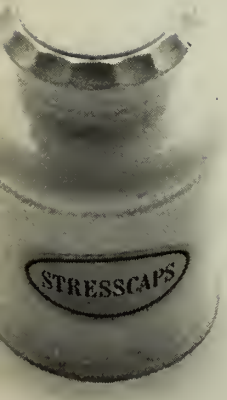
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PREFATORY REMARKS

The wide range of topics presented in this issue of THE JOURNAL-LANCET is illustrative of the breadth of activities taking place in an active pediatric department. Indeed it is representative of the specialty of pediatrics itself which is continually broadening its horizons to meet the health needs of children.

We have attempted to select subjects of interest and value to the physician who cares for children. Of particular significance may be the article entitled "Estimation of Prematurity." In this article is described a clinical research project conducted jointly by a medical student working in the department of pediatrics during the summer months and by an associate professor whose vast knowledge of premature infants was accumulated during years of private and now academic practice. In these days when so much of research seems to demand complex and highly specialized equipment and methodology, it is refreshing to note that careful clinical observations, which may be made by any interested physician, may still contribute significantly to the advancement of medical knowledge.

RICHARD W. OLMSTED, M.D.
Professor and Chairman
Department of Pediatrics
University of Oregon Medical School

Transfusion Therapy in Factor VIII Deficiency

GEORGE W. DANA, M.D.
Portland

THE AVAILABILITY of two new plasma derivatives[1,2] for therapy of bleeding disorders associated with factor VIII deficiency makes this an appropriate time to review transfusion therapy of these conditions. Some theoretical considerations and experience from this institution will be presented.

Among the many disorders characterized by inadequate hemostasis, 4 are currently designated as hemophilia. Hemophilia A (classical hemophilia) is associated with decreased activity of antihemophilic globulin or factor (AHG, AHF, factor VIII). In hemophilia B (Christmas disease), plasma thromboplastin component (PTC, factor IX) activity is deficient. Inadequate plasma thromboplastin antecedent (PTA, factor XI) activity characterizes hemophilia C. The fourth condition designated as hemophilia is variously called vascular hemophilia, pseudohemophilia B, and von Willebrand's disease.

The patients with a familial bleeding tendency described by von Willebrand were not originally recognized as having a defect in any plasma

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TABLE 1
PRODUCTS CONTAINING CLINICALLY USEFUL AMOUNTS OF FACTOR VIII

Product	*Approximate content compared to fresh normal plasma*	
	Albumin Grams per 100 ml.	*Factor VIII per milliliter*
Fresh whole blood	2.75	0.55
Fresh plasma	5	1
Frozen-fresh plasma	5	0.65-0.85
Lyophilized-fresh plasma*	5	1
AHF-rich fibrinogen†	0	1
Cryoprecipitate	0	10
Glycine precipitated anti-hemophilic plasma fraction‡	0	10

*Commerically available as Antihemophiliac Plasma (human), dried, from Hyland Laboratories, Los Angeles, Calif.
†Commercially available as Fibro-AHF from Merck Sharp & Dohme, West Point, Pa.
‡Commercially available as Antihemophilic Factor (human), Hyland Labs, Los Angeles, Calif.
(Significance of the columns to the right explained in the text.)

activity related to coagulation. The only defect disclosed was a prolonged bleeding time. This was ascribed to a capillary abnormality, and the condition was called pseudohemophilia. After Alexander and Goldstein[3] reported the finding of lowered factor VIII in such a patient, a separation was made between those patients with a prolonged bleeding time as their only demonstrable defect and those patients who showed both a prolonged bleeding time and a decreased factor VIII. The latter condition was called pseudohemophilia B. This seems a particularly unfortunate name, as it suggests analogy with hemophilia B and implies a deficiency of factor IX rather than of factor VIII. The name vascular hemophilia does not necessarily try to state etiology, but it certainly carries the connotation that the defect is a capillary one. Reinvestigation of the families on the Äland Islands, originally described by von Willebrand, has shown their bleeding disease to be associated with decreased factor VIII; also, the bleeding time can be corrected by fresh plasma and some plasma products.[4] Therefore, it seems best, at present, to retain the eponym for the hemophilia that has two plasma deficiencies—factor VIII and a bleeding time or von Willebrand's factor.

A discussion of transfusion therapy in factor VIII deficiency, then, will be concerned with classical hemophilia and with von Willebrand's disease. Classical hemophilia will be discussed first and, hereafter, will be referred to simply as hemophilia. The distinction from the other hemophilias has been made only as a reminder that, although the products containing whole fresh plasma activity can be used in the therapy of any of them, the products prepared to concentrate factor VIII will not be effective in treating hemophilia B or C. The terms factor VIII and AHG or AHF activity will be used interchangeably from habit and tradition. Hopefully, this will break monotony rather than produce confusion.

As no other source of factor VIII has been found, plasma or a plasma derivative remain the only products available for replacement therapy in hemophilia. Factor VIII has a short half-disappearance time (eight to twelve hours) in the body and drops rapidly in shed blood. Therefore, the plasma must be used promptly after collection, or its processing, in a way to maintain factor VIII, must be started promptly after collection. The products containing clinically useful amounts of factor VIII are given in Table 1. Considerations in the use of these products can be divided into the medical and the logistical; these are outlined in Table 2. A discussion of all of these considerations is not intended here.

WHOLE PLASMA PRODUCTS

In the control of serious hemorrhagic episodes or protection during surgery, volume has been the most important limiting factor and the prime reason for attempting to concentrate AHG activity. To determine how well the available products meet this first medical consideration, one must take into account: [1] the AHG activity of the product, [2] what level of AHG activity one

TABLE 2
MEDICAL AND LOGISTICAL CONSIDERATIONS IN TRANSFUSION THERAPY OF FACTOR VIII DEFICIENCY

A. Medical
   1. AHG activity in a volume that can be transfused without overloading the circulation
   2. Freedom from side effects
      a. Bacterial contamination
      b. Pyragens
      c. Allergies
      d. Hemolysis
      e. Isoimmunization
      f. Hepatitis
      g. Appearance of circulatory anticoagulants

B. Logistical
   1. Availability when needed
      a. Equipment and personnel for preparation
      b. Storage
   2. Convenience of administration
      a. Frequency
      b. Apparatus
      c. Accessible veins
   3. Cost

wishes to obtain in the patient's plasma, [3] how long one wishes to maintain it, and [4] how long the vehicle carrying the AHG activity will remain in the circulation. As normal plasma is taken as the standard of 100% activity, the percent activity contained in a transfusion of plasma is one hundred times the milliliters of plasma in the transfusion. The effect this can be expected to have on the AHG activity of the recipient's plasma has been given by McMillan and associates[5] according to the formula:

$$AHF_1 = AHF_0 + \frac{100 \times P}{V + P}$$

where AHF_1 = postinfusion plasma factor VIII, activity, percent normal
AHF_0 = preinfusion plasma factor VIII activity, percent normal
P = required fresh plasma, milliliters
V = approximate plasma volume, milliliters (50 ml. per kilogram of body weight).

The part of the formula to the right of the plus sign gives the expected increment (EI). The need for control of hemorrhage is most apt to occur in the severe hemophiliac—one with less than 1% AHG activity. Experience has shown that an AHG activity of 20 to 30% can be expected to control most hemorrhagic episodes. By the use of the formula we would determine the amount of plasma in milliliters per kilogram of body weight needed for transfusion to achieve a 20% rise in activity as:

$$EI = \frac{100 \times \text{ml.} \times \text{kg.}}{50 \times \text{kg.} + \text{ml.} \times \text{kg.}}$$

$$20 = \frac{100 \times \text{ml.} \times \text{kg.}}{(50 + \text{ml.}) \times \text{kg.}}$$

$$\text{ml.} = 12.5 \text{ per kilogram}$$

In this instance, theory and practice go together reasonably well. Most hemorrhagic episodes can be controlled by the use of a "priming" infusion of 15 ml. per kilogram followed by "maintenance" infusions of 10 ml. per kilogram at twelve-hour intervals. How long this program needs to be kept up depends on the nature of the hemorrhagic episode.

Overloading the circulation to the point of heart failure is regularly stated as the limiting factor in continuing replacement therapy with fresh, frozen-fresh, or lyophilized-fresh plasma. The literature is not precise in stating an accumulated amount of plasma that can be infused before this complication is likely to occur. The fact that replacement schedules recommending accumulated daily infusions of 20 ml. per kilogram are limited to seven to ten days suggests that this is before, but near, the point where circulatory failure has been encountered. Or, it may be that authors have found the danger point to be so variable that the recommendation of alertness for the complication is more reasonable than a dogmatic statement as to when it is likely to occur. In a review of the pediatric patients receiving replacement therapy at this institution, just one incident has been found where cardiac embarrassment occurred during the course of therapy.

CASE REPORTS

J. W. was 7 years old and weighed 25 kg. when he was admitted on March 2, 1965, for elective dental extractions. Family history was negative for bleeders; he had been known to have hemophilia since 14 months of age. He was started on transfusions with lyophilized-fresh plasma coincident with the extraction of 4 teeth. He received an average accumulated daily dose of 28 ml. per kilogram. On the twelfth day he showed evidence of cardiac embarrassment with an elevated pulse, a tender liver, an enlarged heart, and a venous pressure of 200 mm. of water. At that time he had received a total accumulated dose of 7,300 ml. of plasma, or 3.7 times his estimated blood volume.

The reason that circulatory overload will eventually occur with continuing transfusions of plasma, any one of which in itself would be inadequate to embarrass the circulation, is that the fluid volume is not removed from the circulation before the next transfusion is given. A greater excess accumulates after each transfusion. The reason that the fluid volume cannot be cleared from the circulation between transfusions, as electrolyte or dextrose solutions would be, is that the plasma proteins with their osmotic pressure are not removed as rapidly as electrolytes or dextrose. Albumin contributes most of the osmotic pressure of the plasma proteins, each gram of albumin in the plasma holding approximately 18 ml. of water with it.[6] Under normal conditions, the half-life of albumin is about ten days.[7] Therefore, it is apparent that, unless there were renal loss or greatly increased catabolism, the addition of 1 gm. per kilogram per day of albumin to the circulation should lead to a continuing expansion of the circulatory volume. If, as another possibility, the albumin load were added to the albumin content of the interstitial fluid, one would expect that eventually fluid distribution (according to the hydrostatic-osmotic pressure relationship hypothesized by Starling) would be disrupted leading to edema and even hypovolemia.

In contrast to the previous case was that of J. G., a 10-week-old infant admitted on August 26, 1965, for investigation of increasing head size, first noted at about 4 weeks of age. A maternal uncle had bled to death following an appendectomy, and two brothers have

proved factor VIII deficiency. His AHG activity was assayed at 4%. Ventriculograms revealed marked hydrocephalus with no air being seen below the lateral ventricules. A right ventriculoatrial shunt using a Spitz-Holter valve was performed. During the period of investigation before surgery and postoperatively, he received from 2 to 6 transfusions of 50 ml. of lyophilized-fresh plasma daily for an accumulated total of 4,600 ml. of plasma over the course of twenty-four days. On the day transfusions were started, his body weight was 6.6 kg., giving an estimated blood volume of 528 ml. In other words, over the course of twenty-four days he received 8.7 times his estimated starting blood volume. At no time did he show evidence of circulatory overload, edema, or cardiac embarrassment. His weight on the day plasma transfusions were stopped was 7.4 kg., and five days later it was 7.3 kg. It did not go below that subsequently, and head size did not increase. One can postulate that, in an infant of this age, renal loss of albumin occurred more readily in the face of an albumin load than occurs in older individuals. Unfortunately, his chart does not record the presence or absence of albuminuria during the period in question.

PLASMA DERIVATIVES

It is the lack of albumin in AHF-rich fibrinogen that gives its main advantage over the whole plasma products. When reconstituted from the dried state, it has the same percentage activity, volume for volume, as do the fresh plasma products. However, because it does not contain albumin, larger amounts can be given in a single transfusion and transfusions can be continued over a longer time. It has been used successfully in the management of surgical conditions for a number of years.[8] A complication that may be seen with AHF-rich fibrinogen, and peculiar to it, is a moderate degree of hemolysis associated with increased rouleaux formation and a positive Coombs' test. This seems to be secondary to the high concentrations of fibrinogen that are reached. Hemolysis is seen more frequently in blood group A recipients than in those who are group O. This can be explained by the concentration of isohemagglutinins in fraction I.[9]

The more recently available products (the glycine-precipitated fraction and cryoprecipitate) have the advantages of low fibrinogen content and concentration of AHG activity in addition to containing no albumin.[2,10] We have had no experience with the glycine-precipitated fraction. It comes in the dried state, each bottle being reconstituted with 30 ml. of distilled water immediately before use. Each bottle is labeled with the number of units of AHG activity that will be contained in the reconstituted 30 ml. Thus, it is a standardized product. A unit of AHG activity is defined as 100% of the activity in 1 ml. of average, normal, pooled human plasma less than one hour old. So, in terms of units, a 200 to 250 ml. donation of fresh plasma would contain 200 to 250 units (if the donor happens to be average-normal in terms of his plasma AHG activity); therein lies the advantage of pooling. This is the range of units supplied per bottle of the glycine precipitate. Thus, the product achieves a ten times concentration of AHG activity in a vehicle that is free of albumin and has a low fibrinogen content. Except for cost ($40 per bottle) and limited production, one would expect it to replace all other products currently available for transfusion therapy of hemophilia.

Cryoprecipitate is the material that does not go into solution when frozen plasma is thawed at refrigerator temperature. After cold thawing and removal of the fluid plasma, the cryoprecipitate is kept in the deep freeze until one is ready to use it. It is then reconstituted in normal saline solution. Cryoprecipitate can be a do-it-yourself product for any blood bank with the necessary time, personnel, and equipment. Prepared according to the method of Pool and Shannon,[1] there is wastage of no part of a whole blood donation, as the remaining fluid plasma is returned to the cells after its removal from the cryoprecipitate. It is not a standardized product. That obtained from each donation is subject to the variation in AHG activity among random donors (50 to 200%), and processing variables are always possible. Pool and Shannon[1] have found in vitro assay of AHG activity of the reconstituted cryoprecipitate not to be an accurate predictor of the rise in activity it produces in a recipient's plasma. (However, the discrepancy was on the favorable side with the effect obtained by transfusion being greater than would be predicted from direct assay of the product.) As direct assay of the product involves entering the container in what is otherwise a closed system up to the time of reconstitution, the chance of contamination is introduced. All in all, it seems best at present to accept it as an unstandardized product and use it only in conjunction with some method for estimating its effect in the recipient. Judging from the response to infusions of pooled lots of cryoprecipitate, Pool and Shannon have found an average of 46% of the AHG activity to be expected in an amount of average-normal plasma from which the cryoprecipitate was prepared. They were able to reconstitute the cryoprecipitate from each donation in a final volume of 13 ml. So, even though recovered AHG activity in the cryoprecipitate was 46% of an average-normal donation (250 to 275 ml.), the fact that this can be reconstituted in 13 ml. means that an approximate

ten times concentration of activity has been achieved ($\frac{0.46 \times 275}{13} = 9.7$).

CASE REPORT

The use of cryoprecipitate is illustrated by the case of R. N. who was 6½ years old and weighed 20 kg. when admitted on January 7, 1966. There have been 9 other known bleeders among his mother's blood male relatives. Two half brothers (same mother) with hemophilia are living, and a third died at 1 year of age from cerebral hemorrhage. Eleven days before admission here, he had entered another hospital because of somnolence, dizziness, headache, and left-sided weakness. Signs of increasing intracranial pressure progressed during the next two days. Nine days before admission here, exploratory burr holes were performed; under the third a large, right temporal, intracerebral hematoma was encountered and evacuated. He became alert following surgery but was semicomatose again within twenty-four hours. The temporal burr hole was reopened, and a hematoma was again evacuated. He did well for seven days following the second operation; then, the same signs recurred. An intracerebral hematoma was evacuated for the third time earlier in the day on which he was transferred to this hospital. Examination here revealed a dilated, fixed right pupil, left hemiparesis, early papilledema, and response to deep pain only. Relief of increased intracranial pressure was obviously necessary.

During the preceding ten days, he had received a single transfusion of 500 ml. of fresh whole blood and then 10 ml. per kilogram of frozen-fresh plasma every twelve hours. This, by estimate, accumulated to 2.5 times his blood volume. Faced with another operation and the desire to maintain AHG activity at at least 20% for another two weeks, it seemed doubtful that this could be accomplished by the use of whole plasma without overloading his circulation. Main reliance was placed on AHF-rich fibrinogen, but the circumstances precipitated our first clinical trial of cryoprecipitate. Three grams of AHF-rich fibrinogen were shown, on one occasion, to bring his partial thromboplastin time (PTT) from a prolonged to a normal value. This was accompanied by a rise in AHG activity from 8 to 26%. This is in line with the observation that a normal PTT can be used to predict an AHG activity of over 20%.

Transfusion with the cryoprecipitate obtained from 2 fresh donations of plasma and processed right away resulted in a rise in AHG activity from 13 to 31%. This 18% rise is a little more than half of the rise that would be expected (33%) had all the plasma from which the cryoprecipitate was prepared been infused and assuming that both donors' plasma contained 100% activity. However, a satisfactory rise was obtained using one-tenth the volume that would have been required to obtain the same rise using "parent" plasma. Also, no albumin was contained in that volume.

On another occasion, the cryoprecipitate used was obtained by simply removing from the deep freeze 4 donations of plasma which had been prepared as frozen-fresh plasma and putting them at 4° C. to thaw. Three of these had been in storage for eight months and 1 for two months. Infusion of the precipitate obtained from these was associated with the return of the PTT from a prolonged to a normal value. Studies on that occasion did not include assay of AHG activity in percentage, so the most one can conclude is that there was a rise from less than 20% to greater than 20% activity. In any event, a satisfactory clinical response was obtained using an albumin-free product with half the volume of the usual frozen-fresh plasma transfusion. It is likely that we would not consistently be this lucky in recovery of AHG activity from aged frozen-fresh plasma, but it does represent a way of getting an albumin-free concentrate when frozen-fresh plasma is available and cryoprecipitate by Pool's method is not. Particularly, it is not recommended for use unless PTT or AHG activity can be followed.

Application of these considerations resulted in a successful outcome as indicated in the completion of the case summary. At operation on the evening of admission here, approximately 60 ml. of mixed blood and necrotic brain were removed from the cavity. Penicillin and chloramphenicol were given, directed against infection for which the area seemed ripe by now. Dexamethasone (Decadron®) was given, also—not for any anticipated effect on bleeding but in the hope of reducing cerebral edema. Evidence of new bleeding had occurred at the other hospital seven days after operation. Rebleeding five to eight days after surgical intervention is a recognized phenomenon in hemophiliacs, presumably associated with dissolution of clots before endothelial repair is complete. In addition to good fortune, infusions containing greater amounts of AHG activity used under laboratory control may account for the smooth recovery of this boy after his fourth operation. No bleeding occurred during the sixteen days during which transfusion therapy was continued after that operation, nor did it occur at the operative site subsequently. Epistaxis occurred eighteen hours after the last transfusion and was controlled promptly by local pressure. Hematuria appeared twenty-seven hours after the last transfusion and cleared in three days without treatment. He left the hospital thirty-three days after admission with the only discernible neurologic deficits being a left homonomous hemianopsia, a disturbance of left lateral conjugate gaze, and a partial abducens palsy on the right.

VON WILLEBRAND'S DISEASE

Control of AHG activity is not as much of a problem in von Willebrand's disease as it is in hemophilia for two reasons. First, the deficiency of factor VIII is less often great enough to present a coagulation problem as it does in hemophilia. Replacement therapy for AHG activity may not be needed and, when it is, will often require a smaller increment. Second, in von Willebrand's disease, transfusion results in increased AHG activity not only by virtue of AHG activity supplied by transfusion but also by appearance of endogenous AHG activity. Endogenous AHG activity does not depend on AHG activity in the transfusion, as it has been reported to follow transfusion of serum, or plasma, from a patient with hemophilia.[4] Aside from the intriguing questions this raises concerning the genetic control of AHG activity, it has the practical consequence that adequate AHG activity can be maintained by transfusion at less frequent intervals.

In addition to factor VIII, control of bleeding in von Willebrand's disease involves consideration of a plasma factor influencing bleeding

time. That this factor is separate from factor VIII has been shown by the correction of the bleeding time in patients with von Willebrand's disease following transfusion of plasma, or a fraction derived from plasma, donated by a hemophiliac.[4] Neither does this factor travel consistently with factor VIII in preparations of normal plasma. In our experience, some transfusions of frozen-fresh or lyophilized-fresh plasma that have been adequate to supply AHG activity have not shortened the bleeding time. This has been the experience of others with these products as well as with AHF-rich fibrinogen.[11]

The bleeding time is a crude test with unexplained variations in normal as well as in affected individuals.[4] This is a nuisance which must be taken into account in using the bleeding time to predict whether or not a patient will have clinical difficulty with bleeding. It also makes one less than dogmatic in stating that a change in the bleeding time is the result of one's therapeutic measures. The Ivy method of determining bleeding time is generally considered to give the more reproducible results, and it is the preferred challenge for diagnostic purposes. When our therapeutic attempts have been accompanied by any change in the bleeding time, a shortening of the bleeding time by the Duke method has occurred without necessarily changing it as determined by the Ivy method. Fortunately, shortening of the Duke bleeding time has correlated well with clinical control of bleeding.

Our limited experience with cryoprecipitate suggests that in this unstandardized product the dissociation of factor VIII and the bleeding time factor can occur in either direction: good AHG activity with little bleeding time factor or good bleeding time factor with little AHG activity. This is illustrated by the following experience with 2 brothers.

CASE REPORT

M. D., a 17-year-old boy weighing 63.4 kg., was admitted on August 29, 1966, for dental restoration and elective extraction of 2 teeth. Family history was negative for bleeders except for 2 affected brothers. He had been recognized to have von Willebrand's disease since age 11 years. He was given the cryoprecipitate from 4 donations of plasma on the day before extractions were done. Immediately before infusion, his Ivy bleeding time was greater than thirty minutes, and his plasma AHG activity was 15%. One and one-half hours after the infusion, his Ivy bleeding time was still greater than thirty minutes and his plasma AHG activity was 60%. As there had been no demonstrated effect on his Ivy bleeding time, a Duke bleeding time was done; it was fourteen minutes. Twenty-four hours later, two hours before extractions were done, his Duke bleeding time was twenty minutes. The 45% increase in his AHG activity had been accompanied by a not-impressive effect on his bleeding time. No more infusions were given as there was no undue bleeding at the time of, or subsequent to, the extractions.

J. D., an 18-year-old brother of M.D. weighing 52.7 kg., was admitted on September 27, 1966, for emergency care of an abscessed tooth. Treatment with penicillin was started immediately, and the tooth was extracted two days later. In the morning on the day of extraction, he received the cryoprecipitate from 3 donations of plasma. Immediately before the infusion, his bleeding time by both the Duke and Ivy methods was greater than forty-five minutes and his plasma AHG activity was 3%. One and one-half hours after infusion, the Ivy bleeding time was still greater than forty-five minutes, 2 determinations of his Duke bleeding time were four and six minutes, and his plasma AHG activity was 16%. Twenty-four hours later, 2 determinations of his Duke bleeding time were two and two and one-half minutes and his plasma AHG activity was 19%. A 13% rise in AHG activity was accompanied by an impressive change in his Duke bleeding time and no demonstrated change in his Ivy bleeding time.

Perkins[12] has reported correction of the Ivy bleeding time in association with infusion of cryoprecipitate into a patient with von Willebrand's disease but has found the bleeding time factor to be more labile than either factor VIII or the factor associated with endogenous factor VIII production. He did not obtain correction of the Ivy bleeding time associated with the infusion of the glycine-precipitated fraction.

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Combined Immunosuppressive Therapy in Steroid-Resistant, Chronic Glomerular Diseases of Childhood

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In 1950 three therapeutic agents, ACTH, corticosteroids, and nitrogen mustard were available for the treatment of the nephrotic syndrome in childhood.[1-3] Both ACTH and the corticosteroids initially proved highly successful. However, a disturbing tendency was noted for some patients to have frequent recurrences and eventually to even become unresponsive to steroid therapy. In others, serious side effects developed if therapy was continued beyond an initial trial.

Nitrogen mustard, by contrast, aroused less interest in the treatment of glomerular lesions. Results of therapy with short-term infusions of mustard were variable.[3,4] Furthermore, nitrogen mustard being relatively toxic required hospitalization of the patient and assiduous care in the details of intravenous administration.

Because of the occasional resistance of the nephrotic state to steroid therapy, combined regimens of corticosteroid or ACTH with mustard were developed.[5-7] Using such an approach in a group of children with lipoid nephrosis who were doing poorly, West reported a very high incidence of diuresis, clearance of proteinuria, and more sustained remissions.[7]

Immunological Basis for Suppressive Therapy

Recent studies have provided new facts about the pathogenesis of glomerular disease. Serial kidney needle biopsies, improved histochemical and immunofluorescent techniques, and application of electron microscopy all have resulted in tentative classification of acute and chronic glomerular diseases into pathogenetic groups. Immunocoagulation studies have permitted clearer understanding of the probable pathological events leading to these identifiable glomerular lesions. Findings resulting from these studies make possible a provisional classification of glomerular lesions according to the following schema (see figure).

A "pure" lesion is that of lipoid nephrosis. Immune complexes in the glomerulus are the exception rather than the rule.[8] Fibrin is not found. Careful examination of the glomerulus via the electron microscope reveals minimal thickening of the basement membrane and smudging of the glomerular epithelial cell foot processes.[9] Lipoid nephrosis appears not to be an immunocoagulation disorder, yet the clinician notes the frequency with which exacerbations follow mild respiratory infections.

Other "pure" types of lesions are found in the toxemia of pregnancy and the hemolytic uremic syndrome.[10-14] In toxemia, capillary endothelial cell swelling and vacuolization are striking. Deposits of fibrin occur between the basement membrane of the glomerulus and the capillary endothelium. A gradual uterine release of thromboplastin in the last trimester of pregnancy in the toxemic patient probably results in partial coagulation.[15,16] Consistently elevated serum levels of fibrin intermediates during toxemia have been reported. Similar experimental lesions have been induced by intravascular clotting and likewise were prevented by the addition of anticoagulants.[17,18] Gamma globulin is not present in these lesions. In the hemolytic uremic syndrome, a histological picture not unlike the Sanarelli-Shwartzman reaction develops.

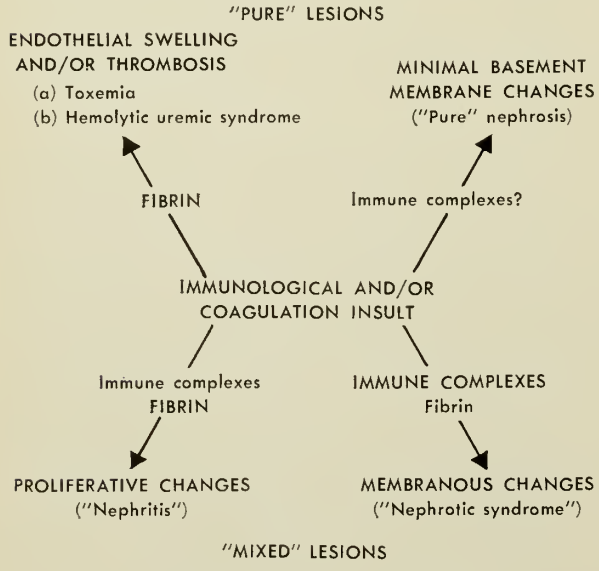
In contrast to toxemia of pregnancy, hemolytic uremic syndrome, and lipoid nephrosis, immune complexes (gamma globulin and complement

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with or without antigen) are almost universally detected in acute glomerulonephritis, lupus nephritis, Schönlein-Henoch's and Goodpasture's syndromes, and subacute states. Immune complexes are also found in chronic proliferative lesions. In chronic membranous glomerulonephritis, these immune complex lesions are marked and may comprise the bulk of the "membrane." Distribution and intensity of complexes in various elements of the glomerulus, that is, the basement membrane and the cells, may be different for different conditions.[9,19-24]

The glomerulus of acute glomerulonephritis, disseminated lupus erythematosus nephropathy, and more severe cases of chronic membranous glomerulonephritis contains fibrin in addition to the immune complexes.[25] Similarly, Schönlein-Henoch's nephritis exhibits material suggesting fibrin.[26] Fibrin is probably a codeterminant, with the immune complex disrupting the normal activity of the glomerulus and exciting detrimental host reactions in these conditions.

It is particularly instructive that fibrin should be found in lesions of hypersensitivity such as acute poststreptococcal glomerulonephritis. Experimental evidence indicates that intravascular coagulation may be brought about by the presence of circulating immune complexes. This suggests coagulation may be an event secondary to immune complex formation.[17] Immune complexes are also toxic to cell membranes, so that one might conclude that the combined effect of cell membrane damage (capillary endothelium of the glomerulus) and intravascular coagulation may be responsible together for the deposition



Polarity of active glomerular lesions

TABLE 1

COMMONLY USED IMMUNOSUPPRESSIVE AGENTS[30]

Agent	*Remarks*
A. Mustards	
1. Mechlorethamine (Mustargen®)	1. Moderate toxicity. Must be given I.V., vesicant
2. Cyclophosphamide (Cytoxan®)	2. Mild toxicity
3. Chlorambucil (Leukeran®)	3. Mild toxicity
B. Purine analogs	
1. 6-mercaptopurine (Purinethol®)	1. Moderate toxicity
2. Azathioprine (Imuran®)	2. Mild to moderate toxicity
3. 6-thioguanine	3. Moderate to marked toxicity
C. Folic acid antagonist	
Methotrexate (formerly amethopterin)	Marked toxicity

of fibrin in such ordinary self-limiting immune disorders as acute glomerulonephritis. Both immune complexes and the fibrin contribute to the final outcome of a given glomerulus.

Theoretically, prevention of immune complexing would seem to have priority over coagulation. Exceptions would include the toxemia of pregnancy and the hemolytic uremic syndrome. Based on the histological evidence found in the hemolytic uremic syndrome suggesting a Shwartzman-like reaction, heparin is now being administered (under controlled conditions) to patients with the hemolytic uremic syndrome at several institutions.[14,19]

While it is clear that the chronic membranous glomerulus is altered by the deposition of large amounts of immune complex, the pathogenesis of proliferative glomerular lesions needs further study. A recent important observation by Vernier and associates[27] that immunosuppression in chronic glomerulonephritis causes regression of proliferative lesions suggests that, whatever the inciting cause, prevention of immune complex formation by immunosuppression may be a key to treatment in steroid-resistant conditions.

IMMUNOSUPPRESSIVE AGENTS

The antiinflammatory corticosteroids have some immunosuppressive effects. Lange and coworkers[28] have shown adequate immunosuppression in a number of cases of disseminated lupus erythematosus. If a treatment response is achieved, continued suppression in chronic glomerular disease may be obtained by the inge-

nious device of alternating corticosteroids.[29,30] Such regimens have reduced the attendant morbidity of long-term therapy on the growing child.

In the cases where corticosteroids fail to alter the progress of glomerular disease, it is necessary to turn to the antimetabolites and alkylating agents, which are also capable of immunosuppression by differential cytotoxic effects on the immune system. These agents may be administered orally and much has been learned about their toxic side effects. Several of these agents have been used in the treatment of chronic glomerular disease (Table 1). Their sites of action and effectiveness have recently been reviewed.[31]

Cyclophosphamide (Cytoxan®), a nonvesicant mustard synthesized in 1958,[32] has cytotoxic potency. Use of Cytoxan® as an immunosuppressant in animals is of interest. The NZW/NZB-F1 mouse has a lupus erythematosus-like nephropathy similar to that in man.[33] Using Cytoxan® to suppress the disease, the following 3 conclusions were drawn on the basis of the study: [1] the syndrome can readily be interrupted with Cytoxan® and, with continuous maintenance doses, apparent permanent arrest of the condition occurs, [2] challenge with an antigen during treatment results in failure of a primary antibody response, and [3] during immunosuppression the animals were not particularly susceptible to infection.[34]

In man, antibody production after administration of an exogenous antigen while being treated with Cytoxan® revealed essentially no humoral response.[35] In addition, Cytoxan® has recently been shown to successfully suppress renal homograft rejection in the human being.[36]

Clinical experience with Cytoxan® is accumulating rapidly (Table 2). In Coldbeck's series, only one therapeutic failure occurred, suggesting case selection was toward early or "pure" lesions in which minimal permanent glomerular damage had occurred.[37] Heymann and Grupe[38] con-

TABLE 2

IMMUNOSUPPRESSIVE THERAPY IN GLOMERULAR DISEASE

Study	*No. of cases*	*Response (%)*	*Diagnosis*
Coldbeck[37]			
Mechlorethamine, cyclophosphamide, and steroids	10	Good, 90 Poor, 10	Steroid-resistant, idiopathic form of nephrotic syndrome, 9 Schönlein-Henoch, 1
Heyman and Grupe[38]		Fair to good, 76	Steroid-resistant nephrosis, 22
Azathioprine or cyclophosphamide and steroids	22	Poor, 10	
Cheng and associates[39]		Good, 50	Lupus, 2
Cyclophosphamide	2	Poor, 50	
West and associates[10,11]		Good, 68	Lipoid nephrosis, 15
Cyclophosphamide and steroids	19	Poor, 32	Hypocomplementemic nephritis, 3 Lupus, 1
U.O.M.S.H. series		Excellent, 28	Chronic glomerulonephritis, 6
Cyclophosphamide and steroids	13	Good, 36 Fair, 27 Poor, 9	Lupus, 2 Lipoid nephrosis, 2 Chronic membranous glomerulonephritis, 3
Lagrue and associates[42]		Excellent, 100 (Complete remissions in all)	Chronic glomerulonephritis, 3
Mechlorethamine, 6-mercaptopurine chlorambucil and steroids	8		Lipoid nephrosis, 2 Lupus, 1 Anaphylactoid purpura, 2
Shearn[43]		Good, 50	Lipoid nephrosis, 3
6-mercaptopurine	10	Poor, 50	Membranous glomerulonephritis, 2 Lupus, 4 Schönlein-Henoch, 1
Vernier and associates[27]		Good to excellent, 65	Chronic glomerulonephritis, 17
Azathioprine and steroids	33	Poor, 35	Steroid-resistant nephrosis, 8 Lupus, 8
Wilson[44]		Good to excellent, 82	Diffuse membranous glomerulonephritis, 3
Azathioprine and steroids	6	Poor, 18	Lupus, 3

TABLE 3

UNIVERSITY OF OREGON MEDICAL SCHOOL HOSPITAL SERIES

Cases	*Clinical and pathological diagnosis*	*Indications for immunosuppressive treatment*	*Duration of leukopenia*	*Response to therapy*	*Side effects*
D.D.C.	Chronic glomerulonephritis	Progressive deterioration, unresponsive to steroids, steroid side effects	8 days	Good	Leukopenia, anemia, and alopecia
M.P.	Chronic glomerulonephritis	Steroid resistance, hypertension, and encephalopathy	4 weeks	Fair	Pancytopenia, alopecia, Klebsiella pneumoniae, and Candida albicans septicemia
L.C.	Chronic glomerulonephritis	Unresponsiveness to steroids, hypertension	2 weeks	Good	Leukopenia, anemia, alopecia, pigmentation
S.E.	Exudative and lobular glomerulonephritis	Biopsy findings plus nephrotic state	2 weeks	Good	Leukopenia, alopecia
M.H.	Chronic membranous glomerulonephritis	Unresponsiveness to steroids, progressive downhill course	2 weeks	Fair	Leukopenia, alopecia
D.L.	Chronic membranous glomerulonephritis	Steroid resistance and side effects	2 weeks	Fair	Leukopenia, alopecia
R.S.	Chronic membranous glomerulonephritis	Steroid resistance and side effects	1 week	Excellent	Leukopenia, mild alopecia
S.P.	Disseminated lupus erythematosus	Steroid resistance and side effects	12 days	Good	Leukopenia, mild alopecia
D.L.	Disseminated lupus erythematosus	Steroid resistance, fulminating lupus	3 weeks	Poor	Leukopenia, alopecia
J.R.	Lipoid nephrosis	Steroid resistance and side effects	2 days	Excellent	Leukopenia, mild alopecia
L.B.	Lipoid nephrosis	Steroid resistance and side effects	4 days	Excellent	Leukopenia, alopecia
D.C.	Focal membranous glomerulonephritis	Preterminal, treated while on dialysis		None	
S.B.	Chronic glomerulonephritis	Preterminal, treated while on dialysis		None	

cluded that corticosteroids improved the response of the patient when used in combination with either Cytoxan® or azathioprine, one being about as efficacious as the other. Although Cheng and associates[39] treated 10 cases of disseminated lupus erythematosus and obtained significant improvement in 7, only 2 had overt nephritis, 1 of whom responded well to Cytoxan® alone. West and associates,[40,41] in their detailed study of the use of Cytoxan® in combination with corticosteroids, reported good overall results in 66% of their patients. Lagrue and his co-workers' results are indeed impressive.[42] One would presume the report was a review of his therapeutic successes, since it is not uncommon for patients to come to medical attention late in their disease.

In the series reported by Shearn, hematological complications of treatment were inordinately severe.[43] It seems likely that such severe bone marrow depressions as described might not have occurred if corticosteroids had been given concurrently. The salutory effect of cortiscosteroids upon hypoplastic bone marrow induced by immunosuppressive drugs is well known, and it may be the most important justification for their use with immunosuppressive agents.

The response to azathioprine and steroids as reported by Vernier and co-workers[27] and by Wilson[44] is probably comparable to the Cytoxan® groups, but the degree of toxicity and the manner of classification of their patients await the detailed published information of their studies.

UNIVERSITY OF OREGON MEDICAL SCHOOL SERIES

While a total of 19 cases of chronic glomerular disease have been treated with combined corticosteroids and Cytoxan®, only 13 are reported here because the period of observation in the other 6 has not been long enough to fully evalu-

ate response to treatment. Excluding the 2 very late, severely azotemic patients dialyzed in order to treat with Cytoxan® and steroids, the favorable results were of approximately the same magnitude as reported by other clinicians. Excellent, in this series, means complete and sustained clearing of proteinuria; good, diuresis, improvement in renal function, and only residual proteinuria; fair, diuresis, but only some reduction in proteinuria, with evidence of gradual progressive decline in renal function; and poor, no measurable response.

Although 3 of our patients fell into the fair category, before combined treatment they had been plagued with almost constant hospitalizations and numerous side effects of their disease and treatment. One adolescent youngster had become suicidal over his crippling disease. They were all moderately azotemic and had hypertension. Each showed improvement as reflected in gradual loss of edema, 1 with clear-cut improvement in renal clearance of urea, and 2 were able to return to school, 1 for seven months and another for a year. These patients were treated early in our series but late in their disease. Improvement in their life situation was considerable while it lasted. None had more than 1 course of combined therapy. With our present knowledge, we would have considered reinstitution of combined therapy for another treatment course. The 2 steroid-resistant lipoid nephrotics and 1 of our chronic membranous nephritis patients gave an excellent response.

CASE REPORTS

Steroid-sensitive nephrosis becoming steroid resistant and then reverting to steroid sensitive again after cyclophosphamide therapy. J.R., 13 years of age, was admitted for the first time to Doernbecher Memorial Hospital for Children at the age of a little more than 8 years for generalized edema and heavy proteinuria following an episode of sore throat and mild fever. Erythrocyte sedimentation rate (ESR) was 32/96 mm., serum cholesterol 725 mg. percent, and total serum protein 3.8 gm. with 1 gm. albumin and 2.8 gm. globulin. Addis count, blood urea nitrogen (BUN), and serum creatinine were normal. Twenty-four-hour urinary protein on admission was 17.28 gm. ACTH, given 100 units daily for ten days, resulted in prompt diuresis. Her urine became protein free nine days after onset of therapy. She was discharged on triamcinolone, 4 mg. four times a day (q.i.d.) given on alternating weeks.

Since then, she has had frequent exacerbations with several periods of complete remission. Steroid dosage was adjusted according to the severity of the disease, and for two years she was quite sensitive to corticosteroids. However, over the period of three years she was admitted 11 times because of nephrotic exacerbations and complications of steroid therapy and gradually became more and more resistant to steroid therapy. Cushingoid features, moderate hypertension, and striae of the abdominal wall developed. Because of repeated therapeutic failures and being teased about being "pregnant," she became quite emotionally depressed. On two occasions she developed the syndrome of nephrotic shock requiring emergency care. During the period of increasing resistance, she developed a duodenal ulcer, incarcerated umbilical hernia requiring surgical repair, peritonitis, and urinary tract infections.

Three and one-half years after onset of her illness, Cytoxan® therapy was instituted. After twenty-one days of treatment, white blood count (WBC) decreased to 3,600 and the drug was discontinued. Prednisone every other day was continued, blood pressure returned to normal, and proteinuria gradually decreased. She was protein free eight months after termination of Cytoxan® therapy. Five months later she again had trace to 1+ proteinuria. Prednisone was gradually increased from 40 mg. every other day (q.o.d.) to 80 mg. q.o.d. with good response. Two years after immunosuppressive therapy she is completely symptom free and urine is negative for protein. Blood chemistries are normal, and growth and development are within normal limits. Sexual maturation is proceeding on schedule, and the patient is menstruating.

Comment. This case illustrates the chronicity of glomerular disease and several serious complications in one child. Rather unique in our experience to date is the transformation from steroid sensitivity to resistance to sensitivity again following combined therapy.

Chronic membranous glomerulonephritis with excellent response to early immunosuppressive therapy. R.S., slightly over 2 years of age, was admitted to Doernbecher Memorial Hospital for Children at the age of 1 year because of generalized edema and proteinuria. ESR was 3/76 mm., BUN 42 mg. percent, serum cholesterol 644 mg. percent, and total serum protein 3.2 gm. percent with 1.1 gm. albumin and 2.3 gm. globulin. Twenty-four-hour urinary protein was 4.3 gm. He was treated with very high doses of prednisone, 60 mg. daily. He became cushingoid and developed hypertension and a monilial infection of the skin. Diuresis occurred only after six weeks of intensive therapy. Proteinuria decreased to a trace and he was discharged on prednisone, 40 mg. q.o.d.

Five days after discharge he developed an ear infection, oral monilial infection, and gastroenteritis. Heavy proteinuria and anasarca recurred unresponsive to high doses of prednisone, intravenous albumin, and diuretics. Paracentesis was required to relieve respiratory distress. Cytoxan® was then instituted after five weeks of unresponsiveness to steroids. There was prompt diuresis. Leukopenia developed after twenty-one days and the drug was discontinued. He was discharged on prednisone, 25 mg. daily. Soon, proteinuria began to fluctuate from negative to 3+. He was readmitted for a renal biopsy which showed moderately severe, chronic membranous glomerulonephritis and nephrocalcinosis. A second course of Cytoxan® was instituted three months after initial combined therapy because of exacerbation. Blood pressure and blood chemistries returned to normal. Proteinuria gradually decreased and disappeared over a period of three months.

Comment. As occasionally occurs, a patient with chronic membranous nephritis may have some initial response to corticosteroids, but he often becomes edematous upon withdrawal of therapy after diuresis and begins to spill protein heavily again. The need for two courses of combined therapy before he became stable and continuously protein free is of note.

Systemic lupus erythematosus with good response to Cytoxan®. S.P., just over 16 years of age, was referred

to Doernbecher Memorial Hospital for Children at the age of 13½ years for evaluation of persistent proteinuria and severe exacerbations of edema and ascites in spite of vigorous steroid therapy since 11 years of age. She was short, cushingoid, hypertensive, and edematous. ESR was 118 mm., serum cholesterol 606 mg. percent, and total serum protein 3.9 gm. with 2.2 gm. albumin and 1.7 gm. globulin. Lupus erythematosus (L.E.) preps were negative (despite a persistent leukopenia), and twenty-four-hour urinary protein determination ranged from 8 to 12 gm. Addis count was markedly abnormal and BUN was normal. Renal biopsy showed changes consistent with systemic L.E. She was unresponsive even to high doses of steroids. Cytoxan® therapy with prednisone, 100 mg. q.o.d., was instituted about four years after onset of her illness. Cytoxan® was discontinued after seventeen days because of leukopenia. Prednisone, 100 mg. q.o.d., was continued after discharge. Proteinuria was gradually decreased from 8.5 gm. to .44 gm. in twenty-four hours over a period of one and one-half years. Blood pressure and blood chemistries returned to normal, and Addis count cleared significantly.

Comment. Until Cytoxan® treatment was undertaken, no clear-cut and stable decline in proteinuria had occurred in a period of two and one-half years. Since combined therapy was instituted, the decline in proteinuria has continued for one and one-half years, showing the prolonged and delayed effect of such treatment.

TABLE 4

REPORTED TOXIC EFFECTS OF CYCLOPHOSPHAMIDE[45]

A. Common	
1. Leukopenia	Intercurrent infection, occasionally during depressed state
2. Nausea, vomiting, anorexia, diarrhea	A moderate reduction in dosage may relieve
3. Alopecia	Occurs almost universally. Hair growth returns while on therapy
B. Less common	
1. Thrombocytopenia, pancytopenia or red cell maturation arrest	
2. Chemical cystitis	Alleviated by temporary reduction in dosage and increased fluid intake
3. Herpes zoster	Apparent activation of latent virus
4. Skin pigmentation	Seen more frequently with chronic treatment
C. Rare	
1. Centrilobular liver necrosis	
2. Hepatomegaly, abdominal pain, and mastitis	
3. Increased platelets	Reported as a rebound with discontinuation of drug
4. Hyperuricemia	A potential hazard in treating renal disease
5. Amenorrhea and teratogenic effects on fetus	

SIDE EFFECTS (Table 4)

Although the toxicity of Cytoxan® is low, certain events in the University of Oregon Medical School series occurred with predictable regularity, namely, alopecia and leukopenia.[45] The alopecia proved transient in all of our patients treated to date. Gastrointestinal upsets have not been a conspicuous finding. Two septic states developed during leukopenia, but in both instances the patient was in metabolic bankruptcy of advanced renal disease. Both responded to appropriate treatment. One child developed a mild case of herpes zoster which occurred a few days after leukopenia was achieved. Pancytopenia occurred in one patient who was uremic but his blood count, which dropped below 300 cells and remained low for one month, gradually improved with corticosteroids and testosterone, as did the platelets. One child developed mild hyperpigmentation.

Toxic effects on the kidney in dogs have been described following massive single doses[46] and with chronic administration in white rats.[47] Bladder toxicity has been reported in children with leukemia or neuroblastomas following prolonged treatment.[48]

TREATMENT PRINCIPLES AND SPECIFICS

With our present limited knowledge, patients who may benefit from combined immunosuppressive therapy fall into the following categories:

1] Patients with lipoid nephrosis who (a) are initially resistant to an adequate trial of corticosteroids, (b) after demonstrating their sensitivity to corticosteroids gradually become resistant over a period of months or years with exacerbations, and (c) develop serious side effects from corticosteroid treatment.

2] Chronic membranous and chronic proliferative nephritides.

3] Progressive subacute nephritides such as may occur with Schönlein-Henoch syndrome or develop following an acute attack of glomerular nephritis.

4] Active lupus nephropathy not readily controlled by intensive suppressive corticosteroid therapy making it difficult or impossible to convert to an alternating steroid program.

5] Trimethadione nephrosis.[49]

An initial detailed renal evaluation should be

done. Additional blood examination should include routine hemogram, ESR, serum uric acid, C-reactive protein, antistreptolysin (ASO) titer, L.E. preps, and SGOT. Chest X-ray and intravenous pyelogram (I.V.P.) (in the absence of severe uremia) should be obtained. If available, antinuclear antibodies, deoxyribonucleic acid (DNA) precipitation test, and complement levels are useful in following the progress of a patient. Should one be in doubt as to the primary renal lesion, a renal biopsy should be considered. In addition to routine histology, such special stains as the Feulgen, methenamine silver, and periodic acid–Schiff (PAS) are an advantage in differentiating lesions which may otherwise defy classification. Specimens for electron microscopy and immunofluorescent examination may be saved for retrospective evaluation of treatment.

Before embarking on a course either of corticosteroids or of combined immunosuppressive therapy, a careful search both by history and by examination for exposure to contagious diseases and chronic sites of infection should be made. An exposure to chickenpox, for example, contraindicates therapy. A positive tuberculin in a child requires careful assessment of the state of activity and isolation and determination of sensitivity of the organism. If the tuberculosis state is recent and active, therapeutic control must first be established. If combined immunosuppressive therapy then seems essential, appropriate antituberculosis therapy should be maintained. Inasmuch as pyelonephritis is more common in association with chronic glomerulonephritides, repeat clean-catch, midstream urine cultures should be obtained as part of the infectious disease screening procedure. Infected patients should be investigated and adequately treated before embarking on immunosuppressive therapy. Prophylaxis should then be maintained during treatment.

If one then still feels committed to a course of combined therapy after the above tasks are completed, an inspection of the BUN, uric acid, and creatinine clearance as well as the patient's general condition, that is, edema and hypertension, will determine the next step.

Milder degrees of azotemia require a calculated reduction in initial immunosuppressive dosage and a more gradual increment schedule, since Cytoxan® and its metabolic products are excreted, in part, in the urine.[50] Moreover, the frankly uremic patient may have hematologic disorders such as anemia and thrombocytopenia and a number of bleeding disorders of diverse etiologies.[51] Unless these are corrected to a significant degree, the use of an alkylating agent is contraindicated. We have dialyzed two children (Table 3) whose uremia otherwise would have made such treatment impossible.

If the resistant patient is very edematous and is threatening skin breakdown, the judicious use of salt-poor albumin in combination with chlorothiazide and spironolactone may result in at least a partial diuresis. Care must be taken to avoid a low-salt syndrome. Scrupulous nursing care of the skin and protection of the lower extremities with such simple devices as a bed cradle can be life saving. If severe edema is refractory to all conservative measures (steroids, immunosuppressives, albumin, and diuretics), peritoneal dialysis with hypertonic glucose will readily remove excess fluid. In most cases the nephrotic child's edema is only mild to moderate and is not a major consideration in determining when to start therapy; however, if the child is rapidly accumulating fluid, a degree of oliguria may occur, and bladder toxicity should be anticipated and the dose of Cytoxan® reduced.

The problem of steroid-induced or potentiated hypertension in chronic glomerular disease may be troublesome. By administering the corticosteroids of the combined immunosuppressive therapy on an every-other-day basis as a single morning dose, hypertension is less difficult to manage. In addition, unless the degree of azotemia contraindicates, adequate control of hypertension can usually be gained by a combination of reserpine and hydralazine hydrochloride (Apresoline®), but occasionally it requires additional agents such as alpha-methyldopa or guanethidine.

TREATMENT REGIMEN

Prednisone in the amount of 2 mg. per kilogram q.o.d. is given as a single morning dose with clear fluids one-half hour before breakfast. In the absence of uremia, Cytoxan® in the amount of 2 mg. per kilogram per day in an oral twice-a-day (b.i.d.) dose is started. Oral prophylactic penicillin, 200,000 units b.i.d., should be given during the course of therapy. Every five days the dose of Cytoxan® is increased by 0.5 mg. per kilogram per day until leukopenia (4,500) occurs.[52] Cytoxan® is then stopped and prednisone is given on a daily basis, 1 mg. per kilogram per twenty-four hours, until the white blood count has returned to 5,000 cells. Leukopenia in this series had its onset varying from seventeen to forty-five days, with an average of twenty-six days. Duration of leukopenia was from two to fourteen days, with an average of eight days. If, however, during the course of combined treatment before leukopenia, the patient has an active

diuresis to dry weight and the urine becomes protein free, Cytoxan® is discontinued. The patient who is not edematous to begin with is monitored by serial twenty-four-hour urinary proteins, Addis counts, and creatinine clearances.

Following the initial leukopenia, should none of the therapeutic end points be reached, it is not necessary to conclude that the therapy is a failure and should be abandoned. Several of our patients have had gradual improvement of renal function as reflected in the quantitative urinary protein over a period of months. A waiting period of six to eight weeks, during which time steroids are continued on alternate days, may allow time for a measurable decline in twenty-four-hour urinary protein output. While monitoring the blood urea nitrogen, biweekly quantitative urinary proteins are satisfactory.

Repeat courses of combined therapy may be necessary before the proteinuria clears completely. In our limited series, repeat courses of treatment do not appear to have obvious cumulative toxic side effects. However, since bladder toxicity has been reported in children treated for neoplastic diseases for long periods of time, hematuria and symptoms of cystitis should be watched for. Adequate fluid intake must be maintained in all patients treated with Cytoxan® to dilute the potentially toxic metabolic products of the alkylating agent.

The striking delay in response does not seem unique to combined immunosuppressive treatment, as Boyd and Commons[53] reported a similar situation in their cases of chronic glomerulonephritis treated with nitrogen mustard. Lagrue and his associates' impressive series, in which an initial course of nitrogen mustard was followed by oral chlorambucil over a period of many months, supports the concept of clearing immune deposits in the more stubborn membranous and proliferative lesions by prolonged immunosuppressive umbrella.

COMMENTS

The use of combined immunosuppressive therapy in an unselected series of patients suggests that about two-thirds will have gratifying responses. With great care in the administration of immunosuppressive drugs, a number of patients who might go on to uremic death are now spared. A group of patients remains who resist combined therapy. It would seem that this group is comprised mainly of late cases with advanced renal damage. Early detection will be necessary to reduce the numbers in this group.

It is tragic to admit a youngster to the hospital in the beginning of adolescence, full of hope and awareness, only to find upon renal biopsy that there is little left but sclerotic or hyalinized glomeruli to work with. The physician has the power to treat. For the many children who do not have routine and semiannual checkups, the physician should consider taking the initiative locally and lead in establishing semiannual or annual automated and computerized screening programs, based on first-voided morning urine samples of school children. With detection, many of the early reversible renal glomerular lesions can be attacked while the physician may still use his drugs effectively.

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(References Continued on page 177)

The Physiologic Basis for the Supportive Management of the Sick Newborn

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A NUMBER OF the important homeostatic adjustments of the newborn infant to illness can be viewed as general patterns of physiologic response to stress. These adjustments are often similar for a variety of different clinical conditions and it is for this reason that it is possible to generalize in part about the supportive management of the sick infant from basic physiologic phenomena. The specific pathophysiology will of course modify these homeostatic patterns of response and the decisions about supportive therapy.

The newborn infant's adjustment to stress takes place within the framework of the body composition[1,2] and energy requirements[3,4] at birth. A greater proportion of the infant's body weight consists of water (72%) than it does in the adult (60%). Most of this extra water is found in the relatively greater extracellular space of the infant. Thus, there is a greater exchangeable sodium and chloride content in the newborn infant than in the adult.[5] The magnitude of these differences from the adult is even greater in the premature infant. Further, the normal pattern of water and electrolyte composition can be altered acutely before delivery,[6–8] making knowledge of the obstetrical history imperative in estimating requirements for the initial supportive therapy of the sick newborn. Overhydration of an anesthetized mother before parturition may result in clinical symptoms of hyponatremia in the infant but not in his mother.[9] And after birth, the stress of severe illness or surgery in the infant will result in excess tissue breakdown with increased production of solutes that must be excreted by the kidneys.

CALORIC REQUIREMENTS

The choice of glucose as the main source of calories for the sick newborn requiring parenteral fluid therapy is firmly supported by a variety of physiologic studies. Glucose is the principal nutrient before birth with fetal blood levels generally fluctuating directly with maternal blood glucose concentrations.[10] In the neonatal period glucose continues to be the principal source of calories but there is a greater utilization of body fat.[11,12] This may impose an additional burden on the premature whose fat stores are smaller at birth. In utero stress, such as asphyxia and extrauterine illness, or premature delivery may decrease normal tissue glycogen stores making specific tissues more vulnerable to injury or resulting in hypoglycemia, or both.[12,13]

Despite the greater body water content, an infant's basic caloric expenditure during the first days of life is double that of an adult on a weight basis (Table 1). The totals presented in Table 1 do not include allowances for calories lost in stool, calories necessary for growth, and the specific dynamic action of food. These are estimated to amount to an additional 50% of basal caloric requirements. Further needs would be imposed by respiratory distress, convulsions, and fever (adding or subtracting 12% for each degree centigrade above or below normal, respectively). The normal maintenance requirements for calories should be based on such estimates of the calories being metabolized and the likelihood of deficits in normal glycogen and fat stores.

The additional caloric needs imposed by illness during the newborn period are qualitatively as well as quantitatively different from those of the adult. The stress of forty-eight hours' fasting in the infant results in much less protein catabolism (4.1%) than in the adult (15.4%).[14] Normally, and probably under stress, the infant meets his caloric needs primarily from carbohydrate and fatty sources if these are available. Thus, smaller loads (10 mOsm per 100 calories metabolized) of solutes for renal excretion (urea, phosphate, and sulfates) result than in the adult (15 to 20 mOsm of tissue solute per 100 calories metabolized). And despite a greater utilization of body

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fat[11] there is less ketosis and ketonuria than in the adult.[15] The protein-sparing effect of carbohydrate which occurs in adults has not been adequately demonstrated in newborn infants receiving comparable glucose supplements.[16]

TEMPERATURE REGULATION

Heat production is a by-product of the consumption of food and its metabolic conversion to kinetic energy. It is closely related to oxygen consumption in the fetus and newborn. In utero the temperature of the fetus is dependent on the production of heat by fetal and placental tissue metabolism and on the transfer of heat between the umbilical and uterine circulations. Fetal heat loss is finely controlled by the well-developed adult circulatory and temperature regulating mechanisms of a healthy pregnant woman. In contrast, the thermal instability and vulnerability of the newborn is related to those factors that determine heat loss to the extramaternal environment. The infant's ability to increase heat production by increased physical activity is not sufficient to keep up with abnormal heat losses. A newborn infant's weight is approximately 5% of that of an adult but its body surface area is about 15% of an adult's surface area. The infant's increased curvatures of body surface and thinner layer of subcutaneous fat (especially in the premature) facilitate heat losses by conduction and radiation. Vasoconstriction is the principal mechanism used by the sick newborn to conserve the heat produced from the increased caloric expenditure. But limiting the environmental heat loss of the newborn is generally more critical and is within the pediatrician's control. Higher survival rates result when premature infants are maintained in an incubator temperature of about 31 to 32° C. rather than 28.5 to 29.5° C.[17] The temperature for maximum survival may be even higher.[18] It seems reasonable to apply such observations to other stressed newborns requiring supportive management although they have not been confirmed by controlled studies in sick newborns suffering from other types of illness. There is evidence that lowering environmental temperature for long periods markedly increases mortality when the infant's body temperature falls to 27 to 32° C.[19] Asphyxia by itself may result in the sustained depression of body temperature.[20] A variety of drugs and anesthetic agents may also lower body temperature.[21]

The failure to maintain the sick newborn's body temperature may result in an increase in heat production and oxygen consumption.[22,23] The heat produced may then be lost to the environment, causing a further reduction in body temperature. During such stress there is an increase in urinary noradrenaline excretion.[24] Hypoglycemia may also be associated with severe hypothermia.[19] The rate of recovery from acidosis is similar in cooled infants and those whose body temperature is maintained at 37° C.[25] However, the effect of cold alone on acidosis is difficult to assess because of the variability of the pattern of peripheral and visceral vasoconstriction which results from cold stress alone in the newborn. The blood sample tested may not be perfusing a tissue that is important as far as a clinical decision is concerned. At present there is no clear-cut pattern of the effects of decrease in the body temperature to the levels frequently seen in sick newborn infants on the function of liver, brain, and kidney in infancy. There is no information available on the cold-induced redistribution of electrolytes and water in the body compartments in sick infants. However, there is ample evidence that moderate hyperthermia alone may result in dehydration and hypernatremia.

TABLE 1

COMPARISON OF FASTING METABOLISM IN RESTING STATE IN HEALTH

	Premature	*Full term*	*Adult*
Basal calories	45-50	40-45	25-30
Activity	7 (15% of basal)	13 (30% of basal)	
Total (minimal)	52-57	53-58	30-50 (sedentary adult)

From Gordon,[3] Levine,[4] and Odell.[5] All estimates are calories per kilogram per twenty-four hours.

WATER AND ELECTROLYTES

The water requirements of the newborn are closely related to his caloric expenditures (Table 2). The amount of water necessary for urine formation is determined in large part by the solutes produced during metabolism that require urinary excretion. Body heat production (calories) results in the insensible water losses from lungs and skin. Such losses can be greatly affected by the environment, for example, insensible water losses from prematures may be 50% greater in room air, resulting in chemical hypertonicity, than when they are maintained in high humidity.[26] The estimate of urinary water requirements in Table 2 is based on the observation that 10 to 20 mOsm of solutes are formed from calories metabolized in a fasting newborn under stress. Odell has pointed out that 60 cc. of urine per

100 calories metabolized would be required to keep the concentration of these solutes safely within the concentrating range of renal function in newborn infants, for example, 167 to 334 mOsm per kilogram.[5] Lastly, the loss of weight itself in the newborn causes a proportionally greater loss of water than solute.[26] This is related in part to the increased extracellular water content.

The smaller losses of electrolytes and nitrogen associated with fasting in normal infants[27] and their increased extracellular water space allow them to tolerate fasting and thirsting during the first few days of life with minimal alterations in body composition under optimal environmental conditions of temperature and humidity. However, the pathophysiologic responses to abnormal antenatal stress or postnatal illness may rapidly exceed the limits of this tolerance and result in alterations in body composition. Under these circumstances the estimated losses are vitally influenced not only by changes in the caloric requirements of the sick infant but by several other physiologic responses to illness which taken together make parenteral fluid therapy a necessity. A moderate degree of illness in the newborn results in considerable increase in tissue catabolism with the consequent formation of a greater solute load for excretion. This makes good renal perfusion and high urine flows a desirable goal in supportive management of the sick infant. Because the glomerular filtration rate is lower in the first few days of life, any further depression has a markedly adverse effect on the total solute excretion. In infants born after prolonged and difficult labors the limited ability of the kidney to excrete nitrogenous waste products may result in a rise in the blood urea nitrogen. The nitrogen production may increase from 79 to 144 mg. per kilogram per twenty-four hours.[28] Prematurity is itself a stress which may double or triple the urinary losses of sodium, potassium, and chloride and increase protein catabolism 30% compared with normal full-term infants under similar conditions of fasting and thirsting. Hansen and Smith found that the renal conservation of water was not only inadequate to prevent hypernatremia at seventy-two hours but there was also a loss of electrolytes and nitrogen.[27] Edematous prematures may have an even greater relative loss of water than electrolytes resulting in more marked hypernatremia.[29] A similar increase in tissue catabolism and renal excretion of electrolytes has been documented in infants with respiratory distress and infants of diabetic mothers.[30]

TABLE 2

WATER AND ELECTROLYTE LOSSES IN NEWBORN INFANTS

	Water (ml. per 100 cal. metabolized)	*Sodium (mEq. per 100 cal.)*	*Potassium (mEq. per 100 cal.)*
Lungs	15 (10- 60)	0	0
Skin	35 (20-100)	0.2 (0.1-3.0)	0.2 (0.2-1.5)
Urine	60 (0-400)	1.5 (0.2-30)	1.5 (0.4-30)
Stool	5 (0- 50)	0.3 (0.1-4)	0.3 (0.2-3.0)
Water of oxidation	−10		

Approximate usual maintenance requirements of infants in first seventy-two hours of life are presented with range of variation. High values represent abnormal losses. Water of oxidation is subtracted since it represents water produced by tissue metabolism. From Nelson's *Textbook of Pediatrics* and Odell.[5]

There is a reduced renal concentrating ability during the neonatal period. Maximal urinary concentration is approximately 600 mOsm. per kilogram of water in the newborn compared to 1,400 mOsm. per kilogram of water in adults. Renal concentrating ability depends in part upon the length of the medullary tubule, which is shorter in infancy, the hypertonicity of the renal papillae, and the urea concentration in renal medullary tissue.[31] The short medullary tubule in the newborn requires a larger volume of glomerular filtrate in order to accomplish solute excretion. The hypertonicity of the medullary interstitial fluid may be decreased by solute losses resulting from stress despite substantial water losses. This may further exaggerate the reduced concentrating ability of the newborn infant. The lessened concentrating ability is probably not due to the absence of antidiuretic hormone or tubular response.[32,33] Dying infants suffering from severe respiratory disturbance and anoxia have markedly reduced urinary excretion of urea and increasing blood urea concentrations due to a combination of their inability to excrete the increased load of solute produced by tissue catabolism[34] and decreased glomerular filtration. Infants with the most marked asphyxia at birth have been found to have the greatest weight losses, largest urine volumes, and most severe hypernatremia at seventy-two hours of life.[35] This hypernatremia is probably further exaggerated by the shift of water into cells that results when there has been significant asphyxia[36] as well as by markedly increased insensible water losses from hyperventilation. Those infants with respiratory distress continue to lose weight after seventy-two hours.[35]

ACIDOSIS

The management of acidosis in the acutely ill newborn is of particular importance. Increased

tissue catabolism results in an increased production of organic acids as well as carbon dioxide. When the particular disease state involves marked muscle activity like convulsions or severe hyperventilation, lactic acid production may be significantly increased with the production of a metabolic acidosis.[37] Prenatally, asphyxia may result in lacticacidemia as well as carbonic acid accumulation due to inadequate placental clearance of carbon dioxide.[38] Postnatally, asphyxia and hyaline membrane disease are the most commonly recognized of a wide variety of illnesses resulting in some degree of mixed respiratory and metabolic acidosis in the newborn. Asphyxia may also result in peripheral[39] or central[13] (depletion of cardiac glycogen) circulatory insufficiency and vice versa. These conditions will exacerbate the acidosis. However, even with milder degrees of illness the circulatory instability of the newborn may play a critical role in the management of acidosis. An increased tissue perfusion may suddenly release acids into the circulation. This can adversely affect myocardial function or pulmonary perfusion, or both. Further, the increased production of organic acids or carbonic acid, or both, may be exaggerated by the renal loss of fixed base which results from the increased tissue catabolism. The sick infant on parenteral fluid therapy may not be able to excrete sufficient hydrogen ions to compensate for the additional acid load, although per unit surface area a young infant taking milk may be capable of excreting hydrogen ions in response to an acid load comparable to the adult because sufficient phosphate buffer is available in the tubular urine. On cow's milk the infant's excretion of titratable acids plus ammonia may exceed the normal adult on a kilogram basis. Smaller and younger premature infants generally but not invariably excrete less urinary ammonia than full-term infants.[40–42]

A clinical evaluation of the degree of general and localized circulatory insufficiency is vital to the interpretation and evaluation of the laboratory analysis of the acid-base status of the blood. A peripheral vein blood sample from a poorly perfused area may exaggerate the degree of acidosis or a central umbilical artery or vein sample from a child with generalized poor peripheral perfusion may lead to an underestimate of the acidosis that will occur as his condition improves and organic acids are picked up from the peripheral tissues.

The body temperature of the infant must also be taken into consideration in appraising the degree of acidosis. A sick newborn's temperature depression may approximate a hypothermic state in which the increased carbonic acid formed by the increased solubility of carbon dioxide is compensated for in large part by the increased blood buffering activity of proteins at the lower temperature. Thus, a pH obtained from an umbilical vein or artery may appear normal at the lower temperature but become markedly acidotic or alkalotic at normal temperatures, depending on respiratory function. The effect on pH of the release of large amounts or organic acids that are accumulating in peripheral tissues will further complicate this situation.

There is the further problem of evaluating laboratory data when the blood is measured at a temperature different from its temperature in the infant. When the infant's temperature is lower than the temperature at which the pH measurements are made, the measured pH will be lower than it actually is in the child. Conversely, if the newborn's temperature is elevated above the 38° C. at which the pH measurements are generally made, his circulating blood will be more acidotic than the blood measured in the pH meter. Estimates of the required alkali therapy must be modified by such clinical considerations.

Lastly, the currently popular Astrup methodology used to determine pH, bicarbonate pCO_2, and base excess is based in part on an assumption that the approximately straight-line relationship between the bicarbonate concentration and the hydrogen ion concentration of the blood in a test tube applies to blood in a patient.[43] There is evidence in adults[44] that when the pCO_2 is moderately elevated, estimates of base excess or deficit based on this assumption may be as much as 4 mEq. per liter off unpredictably in either direction because of the buffering action of extravascular tissues. There is no information available about the error in the Astrup estimates in sick newborn infants. But from studies in adults it seems likely that when either mild metabolic acidosis or acute hypercapnia is superimposed on chronic respiratory acidosis, the determination of pH, pCO_2, bicarbonate, and base excess will not per se distinguish between these 2 complications.[45]

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Phenylketonuria

Report of the Oregon Detection and Evaluation Program

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PHENYLKETONURIA has received world-wide attention over the past ten years. During this time it has become apparent that early detection and treatment can markedly alter the outlook for these children. The purpose of this paper is to describe the detection program and follow-up evaluation in the state of Oregon. It should be emphasized early in the discussion that our series is too small to draw any conclusions. We can only describe our findings and raise certain questions concerning the incidence and natural course of the disease and the completeness of dietary protection. These questions are not original but important enough to bear repeating.

Phenylketonuria was first described in 1934 by a Norwegian biochemist and physician, Dr. Asbjorn Fölling, who discovered the green reaction of ferric chloride when added to urine containing phenylpyruvic acid.[1] Over the ensuing thirty years, the details of the disease entity have begun to unfold. It was discovered that the disease is characterized by an absence, or deficiency, of the enzyme phenylalanine hydroxylase, thus preventing the conversion of phenylalanine to tyrosine. There results an accumulation of phenylalanine in the blood and of certain breakdown products, such as phenylpyruvic acid, in the urine. Phenylketonuria is transmitted as an autosomal recessive and was originally thought to have an incidence of 1 in 20,000.

The classical clinical picture of untreated phenylketonuria has been well described. The children are often fair-haired and blue-eyed with relatively normal development for the first few months of life. There is then a significant change and the infant becomes dull and apathetic and developmental growth slows markedly. Seizures and an eczematoid skin eruption often develop. As time passes, it becomes evident that the child is moderately to severely retarded and often hyperactive with rather characteristic hand play.

In 1951 Woolf suggested a low phenylalanine diet started early in life as a mode of therapy.[2] Three years later Bickel reported the first clinical trial.[3] A 2½-year-old retarded child with phenylketonuria was initially treated with a diet free of, and later low in, phenylalanine content. Serum phenylalanine levels dropped and developmental progress was noted. Regression occurred when the diet was discontinued. Following this initial case, the effects of a low phenylalanine diet were studied further and favorable results were reported.

It soon became apparent that for dietary therapy to be effective, treatment must be started early in life. Attention was then turned towards the search for an inexpensive, reliable test to be used in a mass screening program. In 1961 Guthrie reported that phenylalanine would prevent the inhibition of growth of Bacillus subtilis ATCC 6051 by B-2-thienylalanine in a minimal cultural medium.[4] This bacterial inhibition method has since been adapted for large scale screening programs in many states including Oregon.

Over a decade has passed since the first report of dietary treatment appeared. It has become evident that careful and detailed follow-up programs are essential in order to fully and accurately evaluate results of the low phenylalanine diet. Following is a summary of our program.

The Guthrie inhibition test was first used to screen newborn infants in 1962. Urinary screening became legally a "policy of the state" in 1963. In 1965 the blood test replaced urinary screening. At the present time a Guthrie test is done on heel stick blood before the infant is discharged from the newborn nursery. The mother is instructed by the hospital staff to take her baby to her physician for a repeat blood test within the first six to eight weeks of life. This is done in

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	Group	Age of diagnosis	Age therapy started	Age of evaluation	Upper* extremity motor score	I.Q.	Verbal comprehension score
I							
	1 M.M.	2 wk.	3 wk.	16 mo.		Severe delay	No vocalizaton
A	2 K.P.	7 wk.	8 wk.	16 mo.		Norm. develop.	75%
	3 E.B.	7 wk.	8 wk.	9 mo.		Norm. develop.	100%
	4 J.H.	2 wk.	3 wk.	7 mo.		Norm. develop.	100%
	5 B.R.	4 day	1 wk.	4 yr. 3 mo.	52	47	48%
B	6 M.A.	1 wk.	2 wk.	3 yr. 3 mo.	77	79	77%
	7 P.C.	1 wk.	1 wk.	3 yr.	105	94	84%
	8 H.R.	2 wk.	2 wk.	2 yr. 7 mo.	110	94	97%
II							
	1 D.E.	8 mo.	10 mo.	9 yr. 9 mo.	23	<30	30%
	2 W.T.	9 mo.	11 mo.	7 yr. 9 mo.	66.5	58	65%
	3 T.R.	10 mo.	11 mo.	5 yr. 3 mo.	43	40	32%
	4 S.C.	8 mo.	8 mo.	5 yr. 2 mo.		41	33%
III							
	1 D.B.	2 yr. 6 mo.	2 yr. 7 mo.	4 yr. 5 mo.	75	74	79%
	2 F.H.	2 yr.	2 yr. 2 mo.	5 yr. 1 mo.	34	29	20%
	3 T.H.	15 yr.	18 yr.	22 yr.	Above age for tests	71	Above age for manual
	4 J.T.	17 yr. 9 mo.	——	17 yr. 10 mo.	Above age for tests	77	Above age for manual

*Average for right and left extremities

about 80% of the cases. Since the initiation of the screening program, approximately 30,000 infants have been tested per year with an average detection of 3 new cases per year for an incidence of 1 in 9,000 to 1 in 10,000.

If the initial phenylalanine level is 4 mg. percent or higher, efforts are made to assure a repeat test. If the second serum concentration is 4 mg. percent or greater, repeat tests are requested until the level is normal or until further elevation is noted. If the Guthrie test remains elevated, the blood is assayed by the Turner Fluorometric method for both phenylalanine and tyrosine. The child is considered to have phenylketonuria if the phenylalanine level is 12 mg. percent or higher and the blood tyrosine is not elevated.* The low phenylalanine diet is then started. Initially, serial blood tests may be done every few days or weekly. These are carried out by the private physician or by a public health nurse in the local health department if the physician wishes. Repeat tests are done as often as necessary when the child shows clinical signs of a problem or when serum levels are reported as too high or too low. An attempt is made to keep it between 4 and 6 mg. percent. When the patient has become stabilized, the serum level is determined a minimum of every three months.

It has been the policy of this state for children with phenylketonuria to be followed by their private physicians with the assistance of the director (C.G.A.) and nutritionist of the Maternal and Child Health Section of the Oregon State Board of Health. In addition (in order to obtain more detailed information) each child is referred to one of the authors (J.M.S.) for evaluation and study. The child is then seen within the first year of life for a preliminary examination which includes a pediatric examination, developmental assessment, and language appraisal. At approximately 3 years of age each child is thoroughly examined at the Crippled Children's Division of the University of Oregon Medical School. Detailed evaluation of the child consists of:

1. Pediatric and neurologic examination
2. Dental examination
3. Ophthalmologic evaluation
4. Occupational therapy evaluation: Upper ex-

*Both values are determined to eliminate those instances in which a generalized aminoaciduria is present or those newborns, especially prematures, in whom a temporary elevation of phenylalanine or tyrosine, or both, may be present.

tremity motor test* with items taken from Bayley and Gesell and test for activities of daily living

5. Physical therapy evaluation: Lower extremity motor test with items taken from Bayley and Gesell

6. Speech and hearing assessment: Hearing testing, determination of verbal comprehension, expression, articulation, and intelligibility, assessment of auditory memory, and the administration of the Peabody Picture Vocabulary Test

7. Psychological testing: The Cattell Infant Intelligence Scale, the Vineland Social Maturity Scale, the Stanford-Binet form L-M, and the Wechsler Intelligence Scale for Children

8. Social service interview

9. Electroencephalogram

10. X-rays, including skull film, bone age, long bones, and chest films

11. Urinalysis, gross and microscopic

12. Blood tests: Complete blood count, fasting blood sugar, calcium, blood urea nitrogen, total protein and albumin-globulin ratio, and other tests where indicated.

RESULTS

Sixteen children with phenylketonuria have been seen as part of this study.† They can be divided into 3 groups (Table 1):

Group 1. Treatment started less than 2 months of age, A. Evaluated at about 1 year of age (4), B. Evaluated at about 3 years of age (4)

Group 2. Treatment started at 8 to 12 months of age (4)

Group 3. Treatment started after 2 years of age or no therapy at all, (4).

The results were as follows:

Group 1. Eight children were seen in whom dietary treatment had been initiated under 2 months of age. Of these, 4 were approximately 1 year of age at the time of evaluation.

Two children (Nos. 3 and 4) appeared to be developmentally normal. Patient No. 2 also appeared to be developing normally but showed a four-month language delay. Patient No. 1 has been followed closely by one of us (J.M.S.) and has shown severe physical and developmental delay with possible minor motor seizures. It has been felt that factors other than the phenylketonuria are contributing to her retardation.

The remaining 4 children were approximately 3 years of age at the time they were seen. Two (Nos. 7 and 8) had normal development and no significant abnormalities were noted. Patient No. 6 showed some behavior problems and mild delay which placed him in the borderline intellectual range at present (AAMD classification). Patient No. 5 showed persistent delay in all developmental landmarks and had mild to moderate mental retardation. Repeat psychological testing one year after the original evaluation showed similar results. She also had an abnormally slow electroencephalogram but no history of seizures.

Group 2. There were 4 children, all of whom were started on dietary treatment between 8 and 12 months of age. All of these children had been considered to have had significant developmental delay at the time therapy was started and varying degrees of retardation persisted.

Patient No. 1 has had grand mal seizures and an abnormally slow electroencephalogram. She showed hyperactivity and severe retardation. Two of the children (Nos. 3 and 4) were distractable and moderately retarded and 1 of them (No. 3) has had grand mal seizures in the past but none for four years. Medication had been discontinued. A right frontal spike focus persisted on electroencephalogram. Patient No. 2 showed mild retardation and an abnormal electroencephalogram with slow waves and spikes but no seizure history.

Group 3 consists of 4 children in whom the low phenylalanine diet was begun either after 2 years of age or not at all. Discussion of variation in their clinical pictures follows.

Patient No. 1 was diagnosed at 2½ years of age and treatment started shortly thereafter. He had been hospitalized before therapy was initiated and careful observation and testing showed him to be hyperactive and mildly to moderately retarded. He has shown marked improvement on the diet with behavioral changes and developmental progress. Studies done at the time of his evaluation showed him to be in the borderline intellectual range.

Patient No. 2 was also diagnosed and treated at 2 years of age, but with little change resulting. He was hyperactive and showed severe retardation when seen at 5 years of age.

Patient No. 3 had a period of dietary treatment started at 18 years of age and terminated at 21. This was given in an attempt to better control his seizures. The grand mal seizures have ceased but focal motor seizures persisted. Electroencephalogram showed an abnormality con-

*This test is given to determine the developmental level of hand function of the child with respect to specific grasp patterns and his ability to carry out tasks requiring the finer manipulative skills. Any abnormal patterns of hand use are also noted.

†It should be mentioned that those affected persons living at the Fairview Hospital and Training Center in Salem, Oregon, were not included in this study.

sistent with a seizure disorder. He has had several psyehological evaluations placing him in the borderline range of mental ability. Immaturity and inability to funetion independently have neeessitated plaeement in the Fairview Hospital and Training Center.

The diagnosis of phenylketonuria was made in patient No. 4 when a newborn sibling was shown to have the disease by the Guthrie sereening test. He had been seen by numerous persons for poor aehievement in sehool, borderline intelleet, speeeh problems, and unusual emotional responses, but the diagnosis had not been considered beeause his intelleetual level was much higher than that of the typieal untreated patient. He had reeeived no dietary therapy.

There were no eonsistent abnormalities in the X-rays, blood tests, or urinalyses of these groups.

DISCUSSION

Following the initial report by Bickel in 1954, the low phenylalanine diet became widely used and many reports of its effieacy appeared in the literature. The earliest ones involved the results in older, retarded ehildren who were plaeed on a low phenylalanine diet later in life. Results were variable. In 1955 Armstrong and Tyler[5] reported 5 eases in whom the onset of therapy was from 7 months to 4½ years. Their children became more responsive and seizure control was better. All of the children were initially retarded and, although improvement was noted, some degree of retardation persisted. In 1957 Armstrong and associates[6] mentioned one infant treated at 3 weeks of age with normal development for the five-month follow-up period. Woolf and assoeiates[7] reported similar results in 1958. Hsia and assoeiates[8] in 1958 treated institutionalized ehildren and compared them with affected controls matehed for age, sex, intelligenee, and length of institutionalization, who were given a normal diet. They noted improvement in behavior, rash, and electroeneephalographic abnormalities, but no ehange in intellectual level except in 3 of the younger children.

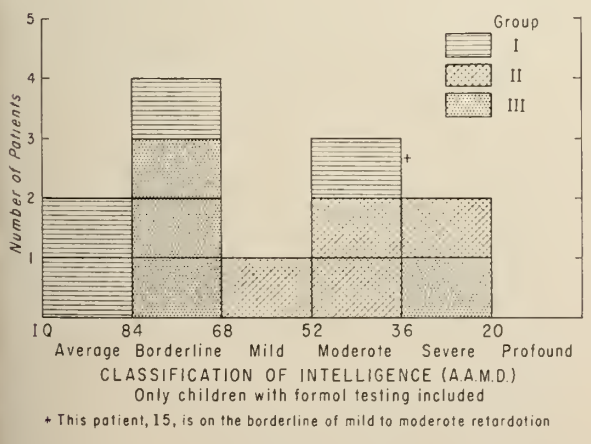


Fig. 1.

In 1959 Horner and Streamer[9] reported in detail 3 ehildren in whom the low phenylalanine diet was started under 2 months of age. The longest period of follow-up was three years. Developmental landmarks were within the normal range for all 3 children. Knox[10] in 1960 summarized all treated cases reported in the literature and concluded that if treatment were started after three years of age there was no significant change in psychometrie performance, but seizures, general behavior, and irritability were improved. He found a signifieant increase in intellectual ability in those treated earlier as eompared to those treated later. This was most marked when treatment was started under 16 months of age. The report of Centerwall and assoeiates[11] in 1961 confirmed this impression but noted that there was some decrease in overall intelligence even when therapy was started quite early and that perhaps the diet did not provide eomplete protection.

The most detailed recent report of ten years of experience is by Sutherland and associates[12] in 1966. In this report all children diagnosed and treated under 3 months of age had normal development. In general those started later did less well. Exceptions to this were apparent, however. Two patients had normal to dull normal intelligenee despite the onset of treatment after 36 months of age. The variability of phenylalanine requirements was stressed.

Our findings, in general, support those of previous reports. The ehildren who were started on the low phenylalanine diet early in life appear to be doing better than those started later. In our small series, however, there are some exceptions whieh bear emphasizing (Fig. 1).

Four of the 8 ehildren in Group 1 (50%) are not functioning up to age level at the present time. In one ehild (No. 1) other faetors may be playing a major role. We fully reeognize the limitations of psyehologieal testing in young children and the limitations of a single test. However, in those instances in which delay was indicated by the intelligence quotient, the delay was eonfirmed by the seores for motor ability in the upper extremities and the scores for verbal eomprehension (Table 1). Eaeh of the 3 examiners ranked the ehildren in essentially the same order (using the test for rank eorrelation). There was no statistical difference at the 5%

level between the mean of the 3 scores. (In calculating these means, only those children were included for whom there were formal scores in all 3 tests.) Thus, the overall impression of delayed development was confirmed independently by several examiners.

Patient No. 5 has been retested and the impression of mild to moderate retardation confirmed. Repeat evaluations will be carried out on all of these children. The important question is why these children are below the expected normal for their age. Two of them (Nos. 1 and 5), who also showed physical growth below the third percentile, had periods of poor intake and low phenylalanine levels during infancy. The significance of this should be considered in view of the recent reports by Rouse[13] on the effects of phenylalanine deficiency on mental development.

Several investigators have questioned whether the diet is completely protective. Centerwall and associates[11] in 1961 and Horner and associates[14] raised the question of damage in utero. It is known that phenylalanine levels may be elevated in the heterozygote pregnant woman[15] and that phenylketonuric mothers can give birth to unaffected retarded children.[16] These facts should be kept in mind when thought is given to further investigation. Consideration should also be given to the emotional effects of this severely restrictive diet on phenylketonuric children. It may be difficult for the parents to think of these children in the same way as their normal siblings when the low phenylalanine diet is so different and may be so difficult to maintain. These problems should be anticipated and met by the physician or team caring for these children.

Three of the 4 children in Group 3 (75%) have borderline intellectual abilities despite late dietary treatment or no therapy at all. Normal or near normal persons with phenylketonuria have been reported[17,18] and the incidence has been estimated to be 1 to 2%.[10] This figure was based on the expected disease incidence of 1 in 20,000. Since the adoption of mass screening programs, however, the incidence of the disease may be 1 in 10,000 or higher. Kleinman[19] questioned the accuracy of the former figure and the true incidence of normal or borderline undetected phenylketonuria. The question can be raised as to whether the increased incidence represents children who would have died in infancy or children who would have been normal or nearly normal and thus escaped detection. Fifty thousand to 100,000 normal school children in the elementary grades should be screened to determine true incidence of the disease in all its various forms before an accurate assessment of the low phenylalanine diet can be made.

SUMMARY

This paper has described the detection and follow-up evaluation program for phenylketonuria in the state of Oregon. Our results have shown that the low phenylalanine diet is the best therapy that we have to offer at the present time. There are many unanswered questions, however, including the reasons for possible incomplete protection by the diet and true incidence of untreated, intellectually normal, affected persons. Further investigation is needed.

The following persons have played an essential role in this evaluation program: Mrs. Florence Scott, nutritionist, State Board of Health; Robert Boyd, Ph.D., psychologist; Doris Bradley, Ph.D., speech pathologist; Virginia Hatch, occupational therapist; Irene Bacon, physical therapist; John Gullickson, D.M.D., dentist; the social workers at the Crippled Children's Division; and Richard L. Sleeter, M.D., director of The Crippled Children's Division.

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Classification and Treatment of Seizure Disorders in Children

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This report will present a simple classification of seizure disorders, with particular attention to their clinical features, comments upon drug therapy, and associated aspects which influence prognosis. The bulk of clinical observations stem from experience with children at the University of Oregon Medical School Hospital and Outpatient Department.

Patients with seizure problems that are more difficult to manage are seen here with greater frequency than by most practitioners. These include the very young, those with mental retardation and other signs of serious central nervous system dysfunction with concurrent behavior problems, and those from homes of limited material resources and capacity for rearing children.

The approach to the management of the child with a seizure disorder is that which obtains in this clinic, and the philosophy underlying it is that presented to medical students and pediatric house officers in the Pediatric Neurology Clinic. This approach is designed to offer, to those who do not often deal with seizures, information which could aid in more accurate classification, increased confidence in diagnosis, and more effective drug therapy. The last-named is the cornerstone of seizure control. Uncertainty of diagnosis can be reflected by reluctance in labeling a child "epileptic" and by hesitancy in prescribing adequate anticonvulsant dosages.

DEFINITION

Nearly a century ago, Hughlings Jackson[1] defined a convulsion as the clinical expression of a paroxysmal discharge from the cerebral cortex. This definition has been amply confirmed but not substantially improved.

The clinical manifestations of such discharge may be abnormal motor activity which is sometimes bizarre or perplexing to an observer, impaired consciousness (all individuals are not necessarily unconscious during a seizure), abnormal or unusual sensations, or complex or simple behavior which is inappropriate to the setting in which it occurs. Combinations of these abnormalities may occur in a particular individual and the pattern, particularly of motor activity, may vary from one seizure to another in a given individual.

The outstanding feature of a seizure disorder is its recurrent and paroxysmal nature. If of unknown cause, it is frequently termed "epilepsy." Any illness or disorder which can affect the structure or function of the brain may cause a seizure. Hence, such seizures are more appropriately termed "the epilepsies."[2] Convulsions of known cause are often labeled symptomatic, or secondary, epilepsy as opposed to "idiopathic epilepsy" of unknown cause. Convulsions may occur in various acute conditions, such as hypocalcemic tetany of the newborn, chemical poisoning, and water intoxication. Conditions not recurrent, or likely to be recurrent, over months or years are excluded from more than passing consideration. Our emphasis will be upon the management of seizures of unknown cause for which there is no specific therapy.

CLASSIFICATION

There is no universally accepted classification of seizures and the physician may have difficulty classifying a particular seizure, whether he observes it himself or, as is more common, as it is described to him by a third party. Accurate classification can be of prognostic value and is necessary for the most effective drug therapy.

The more commonly encountered types of seizures in children are listed.

Grand mal or major motor. This is the seizure commented upon since antiquity.[3] It consists of sudden loss of consciousness, violent tonic-

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clonic movements of the extremities, stertorous respirations, and urinary incontinence. Postictal changes include drowsiness or sleep, headache, confusion, amnesia, aphasia, or paresis of voluntary movement in various combinations. This kind of seizure is described more often in older children and adults than in infants and preschool children.

We have seen several children who presented a seizure pattern which may be a variant of major motor seizures. This begins in the 2- to 5-year age group, but may persist into the second half of the first decade. The seizures are confined to one side, generally involving the arm and leg simultaneously, are tonic and clonic in nature, and last several minutes. Paralysis of brief duration may follow the seizure. The sequence of involvement of the two sides is inconstant, but either side may be involved at any one time.

Petit mal or absence attacks. This is perhaps the most confusing term in the classification of seizure disorders. Petit mal attacks are infrequently encountered in this clinic. The hallmark of the absence attack is the sudden onset and termination of a staring facial expression, seldom of more than thirty seconds duration. Other abnormal activity is usually not noted, although fluttering or blinking of the eyelids, rolling back of the eyeballs, slight tremulousness of the finger tips, or urinary incontinence may be part of the syndrome. Onset is usually after 2 years of age and before adulthood. The electroencephalogram (EEG) typically exhibits 3-per-second spike-and-wave discharge, synchronous in all scalp leads. Robb suggests[2] that the term "absence-simple" be used to describe the least complicated of these spells. The occurrence of repetitive staring spells in an ambulatory patient who is drowsy but is able to avoid stationary objects and speak aloud, though with poor diction, is called "petit mal status."

Staring spells with somewhat different clinical features and associated with other kinds of seizures or EEG findings are probably responsible for the confusion in the application of the term "petit mal." Staring spells of longer duration in a young child with other evidence of central nervous system (CNS) dysfunction are one of the forms of minor motor seizures (see below). Staring spells associated with head turning, lip smacking, chewing movements, or facial pallor or flushing are psychomotor seizures.

Psychomotor. These seizures are uncommonly encountered in the preschool age group. The several patterns of seizures in this category and frequent lack of a complete history can cause the physician anxiety when first confronted with the problem. Abdominal pain with or without nausea, vomiting, or headache often heralds onset of a seizure. This may be followed by a sensation of discomfort rising through the chest into the throat, the so-called "rising epigastric aura." A hallucinatory odor or taste follows, generally described as unpleasant. The seizure may terminate after any of these events or continue with automatic, repetitive behavior, for example, walking around a table or raising a window or more complex, apparently purposeful activity which is inappropriate to the time or place. Some children with this disorder are apparently not unconscious during the complete seizure or part of it. The child may be aware of objects, people, light, or sounds during the seizure but is unable to identify a particular person, recall a sequence of events occurring at the time, or accurately assess the passage of time. Younger children who lack well-developed verbal facility and cannot relate their experiences during a seizure make it difficult to determine whether such a seizure has occurred. Older children may give the impression that they are loathe to talk about their seizures because they regard them with fear, discomfort, or dread. A diffuse and poorly verbalized version may result.

Spike or spike-and-wave discharges arising from the temporal lobe or adjacent areas of the brain may be noted in the EEG. An EEG should be obtained during sleep if the waking EEG is normal.

The following clinical examples illustrate the unusual nature of psychomotor seizures.

Case Reports. Case 1. A 13-year-old boy described recurrent abdominal pain followed by an unpleasant taste. His mother noted staring with lip smacking and concluded, after talking to the boy during and after the seizures, that during the staring spell he was aware of voices, movement of people, and flashes of light but could not accurately identify the voices or people. Upon termination of the seizure he was withdrawn and sullen, usually would not answer questions or obey commands, and, if physically manipulated, would frown, object vocally, and push the manipulator away. This behavior usually persisted for one-half hour but occasionally for up to three hours.

Case 2. An 8-year-old boy would awaken from sleep in the late evening or early morning, get out of bed, and begin putting on his clothes. When asked what he was doing, he would reply that he was getting ready for school. On three occasions, at similar times, he walked downstairs to the kitchen, opened the refrigerator and urinated into it. He was amnesic for both kinds of behavior.

Case 3. A 12-year-old girl experienced frequent episodes of abdominal pain which were followed by facial pallor, a wide-eyed, staring expression, chewing and lip smacking movements, and repeated throat clearing with

expectoration, always into a wastebasket, commode, or sink. She then seemed ill at ease and frightened, for no apparent reason, and was disinclined to answer questions or elaborate upon her symptoms. This girl presented another problem more incapacitating than the seizure disorder. She was unable to maintain pleasant relations with her peers or members of her family. This was accentuated in the postictal state, at which time she was quarrelsome or argumentative with sibs and schoolmates and insubordinate to parents and teachers.

It is of interest that all 3 of these children had irregular spike and spike-wave discharges of paroxysmal nature from the left temporal lobe.

We have been impressed with the fact that the disorder is usually of lesser import to the child and his family than the difficulty the child has functioning well in day-to-day social situations. This has been noted with distressing frequency and has not been readily amenable to treatment.

Minor motor. This group of seizure disorders, in its several forms, is seen frequently in our clinic. The adjective "minor" does not refer to the gravity of the problem but the brief duration of a given seizure.

Myoclonus, or myoclonic jerk, describes a brief, clonic contraction of a muscle or muscle group. Such contractions in the newborn may be considered the equivalent of generalized seizures in older children or adults. The latter seizures are seldom, if ever, seen in the first days or weeks of life with clonic contractions, although tonic seizures may be seen in the neonate. The prognosis of the infant with myoclonic seizures is dismal, as is any seizure in the newborn, in general.

Forceful, generalized flexion of trunk and proximal limb muscles is termed a massive, myoclonic seizure or "infantile spasm." Such seizures are generally first noted between 3 months and 3 years of age. The seizure is of rapid onset and termination and occupies a fraction of a second, although several seizures may occur in rapid succession, especially upon awakening in the morning. The seizure may be missed by an observer unless he is looking at the child when it occurs.

The infant may cry out, usually at the termination of the seizure. The child's mother may be particularly impressed by the cry and flexion of thighs on the abdomen and conclude that the child has "colic"—hence, this frequently noted chief complaint with such patients.

Older children cease to exhibit infantile spasms and are quite likely to develop major motor seizures. Simultaneously, the grossly and diffusely abnormal EEG seen with infantile spasms may become relatively more normal. These changes are apparently a function of brain maturation and independent of earlier seizure control. Death within the first two or three years of life is not uncommon in this group.

Head nodding or head bobbing, either as an isolated event or occurring in flurries, is another form of minor motor seizure which may not be noticed because of its momentary duration. The final category of minor motor seizures is the akinetic or "drop" seizure. These may occur with or without head nodding. They are characterized by a sudden fall from the sitting or standing position. Mothers often relate that the child is very limp if picked up at this time, although an occasional child is rigid or stiff rather than limp. Either way, the episode lasts only a few seconds, and the child reverts to his preseizure state and resumes activity. The child with drop seizures falls many times a day, delivering forceful blows to many parts of his body, notably about the face and head. Broken bones and serious contusions or lacerations are not uncommon.

Children with any form or forms of minor motor seizures are often mentally retarded, either at the time of onset of seizures or after they have been present a few months. Seizures and retardation are not causally related; both are manifestations of underlying, widespread, and serious brain dysfunction which may be progressive or nonprogressive. Effective control of such seizures is not often achieved and usually entails a trial of many different drugs, singly and in combination. ACTH has been used with varying success in the treatment of infantile spasms. Successful control of the seizures does not alter the fact that normal neuromuscular and intellectual development is remote.

FEBRILE SEIZURES

The simultaneous occurrence of fever and convulsion in a child is a vexatious event. The difficulty is highlighted by Robb's statement, "Febrile convulsions present a special problem in epilepsy."[2] Febrile seizures are frequently seen with roseola or shigellosis, particularly in the preschool age group. The mechanism whereby fever causes seizures is unknown.

It seems clear that there is a strong genetic factor in some febrile seizures occurring in otherwise normal individuals. They may be noted in successive generations or in siblings. The management of the problem is well put by Robb: "Like so many problems in medicine, each case must be decided on its own merit."[2] Livingston[4] suggests that a child under 6 years of age whose short (less than five minutes in duration), gen-

eralized seizure occurred a few hours after onset of fever associated with extracranial infection and who has a normal EEG has a simple, febrile seizure disorder, and it is unlikely that he will have nonfebrile seizures. Conversely, the child over 6 years of age who has a focal or prolonged (over 10 minutes) seizure associated with fever and an abnormal EEG is likely to have nonfebrile seizures. There is no way to predict the number of febrile or nonfebrile seizures a given child will have. It is of interest that approximately 50% of Livingston's patients with simple, febrile seizures had only one such seizure.

BREATH-HOLDING SPELLS

Breath-holding spells are unique to the young. Gauk and others[5] have presented the salient clinical features and some interesting physiological studies. The invariant sequence of events is: [1] a provocative event, that is, pain, fright, or frustration followed by [2] apnea, in expiration of thirty to forty seconds' duration, [3] generalized rigidity of skeletal musculature with opisthotonos and extension of extremities, extreme cyanosis, urinary incontinence in some cases, and terminated by [4] a gasp which heralds the return of regular respirations. The child usually resumes his usual activities at this time, although he may be stuporous for a few minutes or hours. Twitching movements of the extremities, of slight amplitude and brief duration, may occur at the time of deepest cyanosis. Frank convulsions are ordinarily not seen. If frank convulsions occur regularly with the breath holding, the child should be considered to have a seizure disorder which is precipitated by the breath-holding spell. The EEG is normal unless obtained during an actual spell, at which time some minor changes may be noted. The outlook is good for spontaneous cessation of the spells. Management consists of calculated neglect by the parent at the time the spell occurs and assurance by the physician that anticonvulsant therapy is unnecessary and that the spells are not damaging to the child. Should frank convulsions occur with the spells, anticonvulsant therapy is indicated. A given child may have only a few breath-holding spells in a lifetime or several a day for months or one or two years. Onset is always below age 5 years, usually before 2, and recurrent spells ordinarily do not occur over a period of two years or after age 5 years.

EVALUATION

The physician's approach to diagnosis and management of a seizure disorder is no different than that toward any other disorder. Often the most difficult problem is whether the behavior described by the historian actually represents a seizure. Much of the history, physical exam, and laboratory determinations is designed to answer this question.

Symptoms developing after the supposed seizure, such as headache, confusion, somnolence, fatigue, or amnesia for the event, suggest that a seizure has occurred. Paralysis or weakness of a part and speech disorder because of dysarthria or aphasia suggest a seizure.

Physical findings reflecting intracranial pathology are asymmetry of form or function of voluntary muscles, unequal growth of bones of the extremities, asymmetry of the hemicrania, or abnormality of transillumination of the skull. The presence of such findings in a child with a supposed seizure history is suggestive not diagnostic of a seizure disorder.

Robb's admonition[2] is well advised, ". . . laboratory tests should be requested with a definite purpose in mind and a knowledge of what useful information may be revealed by the test." Laboratory tests should be undertaken to confirm or deny a clinical diagnosis or distinguish between 2 or 3 diagnostic possibilities. A small-for-gestational-age baby is at risk for the development of hypoglycemia.[6] A prematurely born child who gains weight unusually rapidly on cow's milk and exhibits episodes of apnea may have hypocalcemia. An occasional child is seen with recurrent seizures or movement disorder associated with hypoparathyroidism (hypocalcemia).

The indications for and value of the EEG are sometimes not clear to physicians who encounter seizure problems infrequently. A comment by Robb[2] succinctly described the indications for using this ancillary test: "In patients, particularly children, for whom one cannot obtain a good history, the EEG helps to establish the clinical diagnosis, and sometimes is of value in suggesting a pathological diagnosis. It may be of value as an indicator of the severity of the disorder. This is particularly true in some of the infantile encephalopathies. The EEG is of value in distinguishing hysteria or syncope from a true convulsive disorder. The electroencephalogram should not be considered a means of obtaining a 'penny-in-the-slot' diagnosis. In the first place the test is expensive for the average patient and, secondly, too many tests are ordered unnecessarily before careful consideration is given to what useful information might be obtained. The EEG gives a picture of the electrical activity of the brain as it is reflected through the cortex. It is

rarely diagnostic in itself, but when combined with the clinical history and physical findings may make a complete diagnosis possible."

THERAPY

Those seizures amenable to specific therapy are well presented by others,[7–9] as are the problems peculiar to the neonate.[10] This section will present some general principles of management and comments on a few specific drugs, the use of the latter being the cornerstone of effective medical therapy of seizure disorders. Dosage of a larger array of drugs than presented herein is readily available.[2,4,7,8,11]

The majority of seizure-free patients receive a limited number of drugs. The physician should become familiar with the indications for and dosage of the more commonly used drugs. A knowledge of the effects of overdosage and of untoward reactions which are not necessarily dose dependent is essential.

Treatment is begun with 1 drug only and only 1 drug at a time is added to the regimen or 1 change at a time made in total daily dosage or pattern of administration. Although the time varies with individual metabolism, from ten days to three weeks may be required for maximal blood levels to be reached on a given daily intake. Buchthal and Svensmark[12] have related the blood levels of phenobarbital and diphenylhydantoin (Dilantin®) to clinical control of seizures. The same authors state that, in larger children and adults, minimal variation in blood level is achieved with administration of Dilantin® twice a day and phenobarbital once a day. Children less than 30 kg. in weight may require Dilantin® three times a day and phenobarbital twice a day for the same constancy of blood level.[13] More frequent administration imposes an unnecessary burden upon the mother and increases the likelihood of forgetting a dose. Determinations of drug levels are not readily available, and empirical manipulation of dosage and frequency of administration of anticonvulsants are the usual means of effective seizure control.

The solid forms of medication are preferable to solutions or suspensions. A coarsely powdered tablet or contents of a capsule can be mixed with pureed food, jam, jelly, honey, or peanut butter for the child who cannot, or will not, swallow the intact tablet or capsule. We have found that this results in more accurate drug intake and is mechanically less difficult for the mother. Suspensions, as ordinarily administered, pose a hazard in that they tend to become more concentrated with the passage of time as the active ingredient settles to the bottom. This can result in drug intoxication on apparently moderate doses.

A given drug is increased in dosage until seizure control or signs of overdosage appear. If maximal dosage is without benefit, the drug is discontinued over several days as a new drug is added. If partial control occurs, the minimal effective dose should be administered and a second, third, and so forth drug added and its dose similarly managed. A history of usual or average frequency of occurrence of the seizures, as well as the longest seizure-free interval when not on anticonvulsant therapy, is necessary to determine whether therapy is effective.

Abrupt discontinuation of anticonvulsants, particularly barbiturates, is to be avoided. If the child is temporarily unable to ingest the medication or has vomiting or diarrhea, the medication should be given parenterally. A prescription for a modest amount of the anticonvulsant should be kept on hand in the event the physician cannot be contacted.

Even though the child has been seizure free for several years, it is our practice to continue the medication indefinitely, because cessation of therapy is associated with recurrence of seizures in a significant number of children.

It is not necessary to discontinue the medication before obtaining an EEG on the child. The effect of the commonly used drugs on the EEG is known by competent electroencephalographers, is seldom of a gross nature, and will not interfere with interpretation of the record.

Therapy is a family affair and the responsible members of the family, and the patient when possible, should know the technical or proprietary name of the drugs used, the frequency of administration, and the size of dosage form and should be aware of signs of overdosage.

Phenobarbital can be used as the initial drug in the treatment of any kind of seizure. The best results are obtained with major motor and some absence-simple spells. The frequency of absence spells may increase with barbiturate administration and cannot be predicted beforehand. If seizures increase, some other medication is employed. Phenobarbital sometimes causes hyperactivity, irritability, or other mood disturbances. Changing to mephobarbital (Mebaral®) or metharbital (Gemonil®) has usually produced improvement in behavior without altering seizure control. The reason for this is unclear, particularly since Mebaral® is converted into phenobarbital after ingestion.[14]

Moderate doses of phenobarbital may cause drowsiness for a few days, and the physician's statement to this effect will avoid unnecessary

anxiety for some parents. The word "phenobarbital" may have unfavorable connotation for some parents, but a simple statement by the physician indicating the safety of barbiturates at the time they are prescribed may prove of great value to parent and physician. The rate of metabolic degradation of Dilantin® may be accelerated by phenobarbital.[15] On occasion, this may require an increase in the daily dosage of Dilantin®.

The indications for use of Mebaral® and Gemonil® are similar to those of phenobarbital. Phenobarbital is probably the safest and most effective single anticonvulsant. Overdosage is manifested by drowsiness, incoordination, and, in some children, untoward mood or behavior. A reasonable initial dose is 3 to 4 mg. per kilogram per day given at bedtime. Twice this amount of Mebaral® and one and one-half to three times this much Gemonil® are comparable starting doses.

Acetazolamide (Diamox®) is a safe drug and may be used in all forms of seizures but appears to be of greater benefit with absence-simple or minor motor seizures. It does not produce mood disturbances or hyperactivity as a rule. It is relatively expensive and may produce dryness of the mouth, anorexia, infrequent urinary incontinence, and mild weight loss. Our practice has been to administer 125 to 250 mg. at a time, two to three times daily as a starting dose. We have seldom found it necessary to exceed 750 to 1,000 mg. per day.

Primidone (Mysoline®), in a dosage of 125 to 250 mg. two to three times a day, is used in the treatment of major motor and psychomotor seizures. It is a safe drug, but the anticipated, full daily dose must be achieved by gradual increase in daily dosage over a period of several days. Immediate administration of full, daily dose may produce profound depression of consciousness.

Dilantin® is used in the treatment of major motor or psychomotor seizures. It is of little value in absence-simple or minor motor seizures. A very occasional patient with the latter problem may exhibit noticeable (to the mother) improvement in mood or behavior. It is a relatively safe drug, although a variety of untoward effects have been reported. Hirsutism and gum hypertrophy, apparently not dose related, may occur. A small number of our patients have been changed to ethotin (Peganone®), 250 to 500 mg. two to three times a day, with no adverse effect on seizure control and disappearance of gum hypertrophy and hirsutism. An average starting dose of Dilantin® is 5 mg. per kilogram per day administered in 1 or 2 doses.

Trimethadione (Tridione®), 150 to 300 mg. two to three times daily, is an effective drug for absence-simple attacks and is also of value in minor motor, but not major motor or psychomotor, seizures. It is seldom necessary to exceed 1.8 to 2.1 gm. per day for effective control of seizures. Higher doses may depress the CNS.

The indications for the use of Tridione® also apply to ethosuximide (Zarontin®). A common initial dosage is 125 to 250 mg. two to three times per day. We prefer the use of Zarontin® because it appears to be relatively safer.

Administration of either Zarontin® or Tridione® requires at least monthly urinalyses, white blood count, and differential count, since either drug may depress bone marrow function or cause renal injury.

This is not an exhaustive list of anticonvulsants, but familiarity with this group of drugs will provide the physician with the basic tool for maximal effective therapy of most seizure disorders.

STATUS EPILEPTICUS

This is a hazardous condition and requires emergent treatment. A modification of the treatment presented by Yannet[16] has been employed for some time at this hospital. Treatment is begun when the child is first seen. Sodium phenobarbital (8 to 10 mg. per kilogram) is given intramuscularly immediately. If the seizure has not terminated in twenty to thirty minutes, half the initial dose is given intramuscularly, and this is repeated every twenty to thirty minutes until the seizure is reduced to sporadic contractions of a muscle or muscle group. The lower dose is used for seizures of shorter duration, thirty to sixty minutes, that are clonic in nature or when an anticonvulsant has recently been given elsewhere and the amount and identity, or both, of the drug are unknown.

Intravenous phenobarbital may be used if the physician is experienced in its use and if frequently checked equipment and an anesthesiologist are immediately available in the event that respiratory depression develops.[17]

The intramuscular route has been found to be very effective and, provided the indicated dose is given, it is very safe. Most seizures are controlled with 12 to 15 mg. per kilogram of phenobarbital being given over twenty to forty minutes. We have experienced no respiratory depression with this method of treatment, there have been no fatalities, and only one case did not respond well. This was in the case of a 10-kg.

child who required 1.8 gm. of phenobarbital intramuscularly and intravenously over thirty-six to forty-eight hours before seizures were controlled.

PROGNOSIS

The prognosis of seizure disorders depends upon many factors, all of which are not known. As a rule, the younger the child at the time of onset of seizure, the more likely CNS disorder is present and the more difficult seizures are to control. The presence of mental retardation or an abnormal neurological exam usually indicates more difficulty in achieving seizure control. Conversely, the older child with normal intelligence and a normal physical exam is a better candidate for complete seizure control.

Individuals with a strong family history of major motor seizures have a relatively optimistic prospect for good control.

Children with infantile spasms or minor motor seizures have a grim prognosis for normal intellectual and neuromuscular functioning, even though the seizures may be controlled.[17] Our success in control of the former type of seizure, particularly, has been less effective than that reported by some.

Our limited experience with absence-simple spells does not warrant generalization, but outlook is good for complete control or significant reduction in seizure frequency. Major motor seizures occur in a significant percentage of these children, before or after onset of absence.

Psychomotor seizures have been difficult to control as have the behavioral and social problems associated with the seizures. Associated major motor seizures are another commonly encountered handicap in this group.

The control of seizures is not always simply and quickly accomplished. A first approach consists of classification of the seizure within rather broad categories, administration of an appropriate drug, and observation for change in seizure frequency and undesired side effects of the drug. Subsequent management may require empiric adjustment of drug and dose and considerable dialogue between physician and family. The goal of therapy is complete control of seizures in a normally functioning child. This requires a joint effort between the child's family and physician and success is well worth the time, effort, and money expended.*

*The monograph prepared by Robb is available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C., 20302, for 45 cents. It is a concise and lucidly written survey of the current status of knowledge of epilepsy and is highly recommended.

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Determination of Gestational Age in Premature Infants

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BIRTH SIZE as a measure of the duration of gestation is quite inadequate. The traditional definition of "prematurity," indicating an infant of 2,500 gm. (5½ lb.) or less at birth, has been replaced by the term "low birth weight," since more than one-third of infants in this weight category (2% of all newborns) are actually of term gestation. Conversely, an approximate 3% of all infants with a birth weight over 2,500 gm. have a gestation of less than 37 weeks.[1]

Knowledge of the last menstrual period is an important measure of probable gestational age. However, the irregularities of menstruation and the failure of many women to record this event leave a significant number of infants with an indefinite period of gestation. Other parameters which have been utilized to separate the mature from the immature baby at birth have been physical observation,[2] desire for food,[3] neuromuscular maturation,[4-7] metabolic activity,[8,9] and response to photic stimulation.[10]

Regardless of the methods employed, the degree of prematurity is not easily determined at one observation. However, by recording the longitudinal achievement of certain neuromuscular "milestones," a pattern of development unfolds which will place the infant's maturity in relation to his postmenstrual age. (Postmenstrual age refers to the interval between the onset of the mother's last menstrual period [since exact time of conception is often unknown] and the day of observation of the infant, or the gestational age plus the postnatal age.) The following study was designed to observe this maturation by periodically testing infants with five easily performed procedures which assess the neuromuscular state.

SAMPLE

Thirty-one white infants, 15 males and 16 females, compose this sample. They were admitted to the study free of disease or abnormality and had a reasonably well-documented last menstrual period. The gestational ages averaged 31½ weeks. Their birth weight fell between the tenth and ninetieth percentile of the Lubchenco grid for their gestational age.[11]

METHOD

Each infant was observed at weekly intervals to determine his neuromuscular development, indicated by his response to five different procedures as outlined below. The infants were awake and tested before feedings.

	Technique	*Successful response*
1. Knee flexion (Fig. 1)	Place in prone position with legs stretched out	Full knee flexion with or without raised hips
2. Suck	Place pacifier or nipple bottle in mouth	Nipple sucking sustained for at least 5 sucks or completion of an appropriate feeding in ten to fifteen minutes, or both
3. Moro reflex, grasp component (Figs. 2A and B)	Support head and back at 45° of inclination. Allow head to drop back 30°	Flexion of arms at elbows without flailing
4. Arm traction (Figs. 3A and B)	Lift infant from supine position by forearms to a point of 45° inclination	Maintenance of head nearly parallel to trunk (best of 3 trials)
5. Head extension (Fig. 4)	Place infant in prone position with head in midline, flicking buttocks or feet for stimulation	Raising of head off the bed (best of 3 trials)

Observations in the immediate newborn period (forty-eight hours) or at the time of illness may give an unsatisfactory response.

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Fig. 1. The successful response of lateral knee flexions

RESULTS

Figure 5 shows the postmenstrual ages at which the developmental "milestones" were reached.

1] Knee flexion occurred at 31 ± 1 weeks of postmenstrual age in 72% of those tested.

2] Effective suck for formula requirements occurred at 33 ± 1 weeks of postmenstrual age in 97% of those tested.

3] The embrace component of the Moro reflex was achieved at 35 ± 1 weeks of postmenstrual age in 82% of those tested.

4] Neck flexion traction developed at 36 ± 1 weeks of postmenstrual age in 68% of those tested.

5] Neck extension in the prone position occurred at 37 ± 1 weeks of postmenstrual age in 84% of those tested.

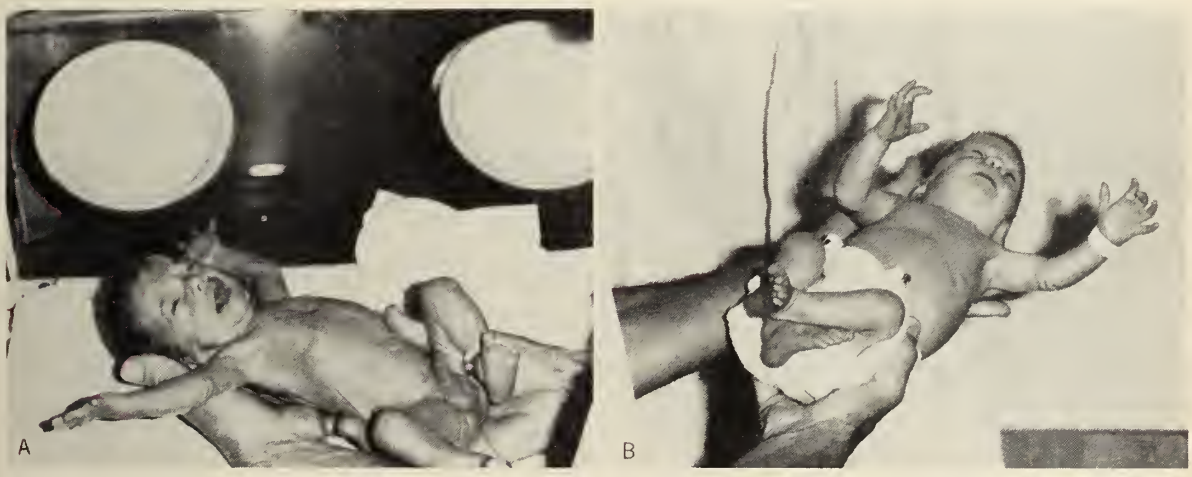


Fig. 2. [A] The lateral component of the Moro reflex. [B] The "embrace" or mature component of the Moro reflex. This should be graded as mature only when the infant flexes his forearms bilaterally in the embrace. Superior extension and flailing should not be evaluated.



Fig. 3. [A] Inadequate or immature neck flexion on traction. [B] Adequate or mature neck flexion on traction



Fig. 4. Mature head extension when placed in the prone position. The infant must extend his chin off the mattress to be considered passable. The infant moving his head only to the side is not passable.

COMMENTS

Neuromuscular maturation parallels fetopostnatal age, irrespective of the time of birth.[5] The longitudinal observations used in this study easily show this progression in maturation. Thus, an immature infant of unknown gestation who attains these responses in a sequential manner will suggest his postmenstrual age. Subtraction of his postnatal age from the mean neurologic age, as assessed by achievement of specific response, gives an approximation of his true gestational age. Failure to follow reasonably this developmental pattern may indicate neurologic disease, cerebral injury, or maldevelopment.

The longitudinal neurologic assessment of the prematurely born infant in judging maturity has been reported by others.[4-7] Although the development of reflex behavior and neuromuscular tone does not always lend itself to precise timing, the emergence of the five responses used in this study could be timed with reasonable accuracy.

SUMMARY

Thirty-one white, immature infants of known gestational age were longitudinally observed for the time at which certain neuromuscular responses were achieved. The responses recorded were knee flexion, effective suck, embrace component in the Moro reflex, and head control in neck flexion and extension.

These "milestones" in development occurred at

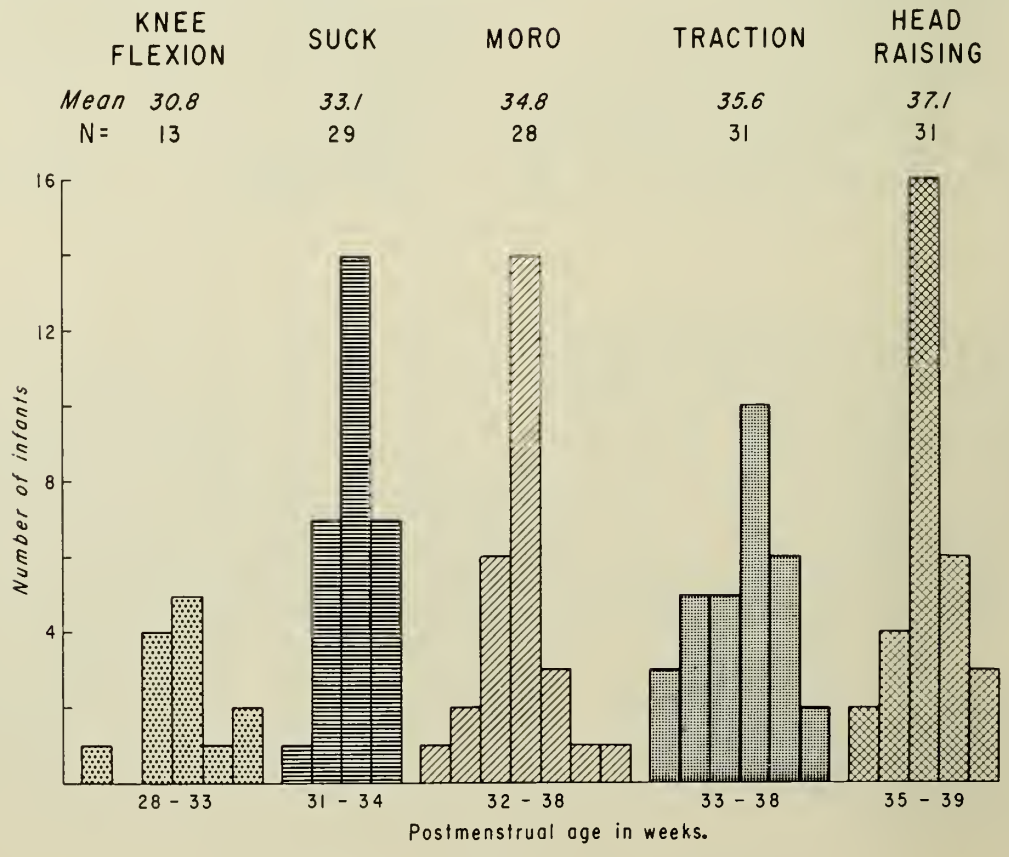


Fig. 5

fairly definite intervals in relation to the infants postmenstrual age (gestational plus postnatal), regardless of the postnatal age of the infant or the degree of prematurity of the infant at birth. Recording the time at which these positive responses were observed should give a retrospective approximation of gestational age in infants born from mothers with an unknown last menstrual period.

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Continued from page 156

COMBINED IMMUNOSUPPRESSIVE THERAPY IN STEROID-RESISTANT, CHRONIC GLOMERULAR DISEASES OF CHILDHOOD

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Heart Failure in Infancy

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When heart failure occurs in infancy, it is usually the result of structural abnormality. Most infants who are predestined by the severity of their lesion to develop heart failure will manifest this complication within the first six months of life. Thus, it is unusual for the initial onset of heart failure to occur in an older infant or child unless there is a complicating factor, such as anemia, arrhythmia, bacterial endocarditis, rheumatic fever, or acute glomerulonephritis. Heart failure also may follow heart surgery, especially if a ventriculotomy is involved.

GENERAL CONSIDERATIONS

About 20% of infants born with congenital heart disease will develop heart failure.

Most of the failure-producing lesions affect the left heart initially,[1] but right-sided failure may ensue as a result of pulmonary hypertension.

When there is a severe obstruction to the flow of blood, heart failure occurs early in life, often within the first week. Such lesions include the hypoplastic left heart syndrome (mitral atresia, left ventricular hypoplasia, aortic atresia, and hypoplastic aortic arch in various combinations), some types of total anomalous pulmonary venous return, and severe aortic stenosis.

By contrast, when there is a communication between the left and right heart (e.g. patent ductus arteriosus or ventricular septal defect), left heart failure generally occurs later—typically in the third or fourth month of life. This delayed onset probably reflects the high right-sided pressures in the newborn, preventing the shunting of blood across the defect and thus avoiding volume overload of the pulmonary circulation.

In severe isolated pulmonic stenosis and preductal coarctation, the burden falls primarily on the right heart, and isolated right heart failure may be seen. Transposition of great arteries may be associated with failure of either side or both.

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SYMPTOMS AND SIGNS

Parents usually will mention feeding difficulty, poor weight gain, inappropriate sweating, and labored or rapid breathing as their major concerns. Often, such infants become exhausted after a few minutes' feeding. Undernutrition frequently results from an inadequate food intake and may be augmented by a high rate of oxidative metabolism relative to body weight.[2] Excessive sweating is common and may be the result of increased catecholamine production. Dyspnea and tachypnea are the consequences of left heart failure. Sometimes wheezing may be the primary symptom and the major auscultatory sign. It is easy to mistake the clinical picture for bronchiolitis, but wheezing is the result of alveolar and bronchiolar transudate rather than being of inflammatory origin. Peripheral edema may be evident as puffiness around the eyes or backs of hands or feet. Serous effusions are unusual but usually indicate a rapidly deteriorating infant.

The most useful physical signs of left heart failure are tachypnea, pulmonary rales, and rhonchi. Right heart failure is manifested by liver enlargement and, occasionally, peripheral edema. Observation of the neck veins for estimation of venous pressure is difficult. In either left- or right-sided heart failure, the heart rate is usually increased to 120 to 170 beats per minute. A gallop rhythm or pulsus alternans occasionally is observed, especially with myocardial disease.

Symptoms and signs of heart failure may make it difficult to verify underlying structural heart disease. However, usually there is an organic-sounding murmur or obvious central cyanosis.

Whenever heart failure is suspected, an electrocardiogram and chest X-ray are mandatory. Thus, the electrocardiogram may reveal the ST-T wave changes of myocardial disease or the myocardial infarction pattern almost diagnostic of anomalous origin of the left coronary artery. Similarly the X-ray may reveal unsuspected cardiomegaly or abnormality of contour.

MANAGEMENT

Once the diagnosis of heart failure has been established, there are two alternatives: [1] re-

gard the infant as an emergency with immediate hemodynamic study and angiocardiography or [2] allow a period of intensive medical therapy before considering these investigations.

Fortunately, the correct course is usually determined by age. The 1- to 7-day-old infant with heart failure usually deteriorates steadily. His downhill course may be slowed by medical therapy but effective surgical intervention based on anatomic and hemodynamic understanding of his malformation generally offers the only hope of survival. Thus, aggressive medical treatment should be started immediately and cardiac catheterization and angiocardiography should usually be performed within a few hours of hospital admission. If the defect is operable, immediate surgery may be a life-saving procedure.

A classic example of this situation is the infant with severe pulmonic stenosis or pulmonary atresia with intact ventricular septum whose only effective pulmonary blood flow is supplied by a patent ductus arteriosus. In such infants, the ductus often closes on the third or fourth day of life, resulting in death. If pulmonary valvotomy can be performed before this happens, the outlook is excellent.

By contrast, the infant who develops heart failure for the first time at 2 to 6 months of age usually can be helped by medical measures. Cardiac catheterization, if indicated, can be performed electively a few days after admission.

When heart failure is encountered, one should be alert to the possibility that no structural heart disease is present. Thus, paroxysmal atrial tachycardia (PAT) or Coxsackie myocarditis may occur in an otherwise normal infant and often leads to heart failure.

The medical management of the infant in heart failure may be conveniently divided into those measures which should be considered for all and those which are ordinarily reserved for life-threatening situations.

Routine measures. 1. Thermal environment. The infant in heart failure is best observed clothed only in a diaper and in a "neutral" thermal environment, that is, a temperature at which the infant's resting oxidative metabolism (and thus his circulatory needs) is minimal. For the neonate, this is 88 to 90° F. and for the 6-month-old undernourished, and often hypermetabolic, infant, 78 to 85° F. For neonates this is best achieved with an incubator in which the baby is clearly visible from across the room. Environmental temperature can be manually controlled or, if the incubator is equipped with a "skin thermistor servo-control," the infant selects whatever temperature is necessary to maintain skin temperature at 97° F. Too warm an environment will cause fever and thus increase resting metabolism. Too cold an environment will cause increased metabolism to generate more heat. The ideal "neutral" air temperature will lie between 78 and 90° F., depending on age of the infant.

2. Posture. Young infants should lie on a 20 to 30° sloping incline inside the incubator. This allows venous blood to pool in the legs and probably decreases the work of breathing by lessening the compression of the diaphragm by the abdominal contents. If the slope is steeper, it may be necessary to put tape from the axillae to the mattress. Older infants may be nursed conveniently in an infant seat.

3. Feeding. Infants who tire easily often benefit by feeding every three or even every two hours. Extremely sick infants find even short periods of feeding difficult because of dyspnea, and they may aspirate milk. Gavage feeding overcomes this problem. A low-salt milk, such as Lonalac®, may be used for a few days but there is a danger of salt depletion with continued use. A good compromise is a milk low in mineral content, such as Similac® PM 60/40. Increasing the concentration to 25 calories per ounce results in increased caloric intake in many instances.

4. Oxygen. The infant with heart failure may benefit considerably from breathing an oxygen-enriched atmosphere, generally 30 to 40%. Even if pulmonary-venous and arterial blood are fully saturated, additional dissolved oxygen can be carried in physical solution in plasma, thus decreasing the circulatory needs by a small factor.

5. Treatment of anemia. The oxygen-carrying capacity of blood will be increased by treating anemia. The optimal hematocrit for the acyanotic baby with heart failure is probably between 40 and 45%.

6. Heart rate. Optimal heart rate is probably 100 to 140 beats per minute. The infant with heart failure often has a heart rate of 160 to 170. This tachycardia responds to control of the heart failure but certain arrhythmias require specific treatment, notably paroxysmal atrial tachycardia (PAT) and atrial flutter.

7. Digitalis. The infant with heart failure should receive digitalis in every instance unless the heart rate is below 100 per minute. Many preparations are available, but we prefer digoxin (Lanoxin®), which is available as a pediatric elixir and a parenteral solution. The twenty-four hour digitalizing dose will depend on the age and maturity of the infant (see table). The newborn and, especially, the premature newborn are extrasensitive to digitalis. Any suspicion of digi-

SCALE FOR ADMINISTRATION OF DIGITALIS OVER 24 HOURS

Age	*Digitalizing dose of oral digoxin (mg. per kilogram)*
Prematures, up to 2 weeks	.03
Prematures, 2 weeks to 3 months	.04
Term newborn, 0 up to 1 week	.05
Term newborns, 1 week to 3 months	.07
Infants, 3 months to 1 year	.08
Children, 1 to 3 years (20 to 35 lb.)	.06
Children, 3 to 9 years (35 to 80 lb.)	.04
Over 9 years	Consider surface area and relate to adult dose

Parenteral dose should be two-thirds of oral dose; maintenance, one-fourth to one-third of digitalizing dose per day. Individual tolerance varies at least twofold. This schedule will be safe in at least 95% of the cases, but under electrocardiographic control, increases will he needed in many instances.

talis intoxication (arrhythmia, tachycardia, or bradycardia) is an indication for an electrocardiogram and possibly withholding the drug.

8. Diuretics. Meralluride (Mercuhydrin®), 0.1 ml. per 10 lb. of body weight, or thiazide diuretics are sometimes indicated. However, there is a real danger of potassium depletion which potentiates the action of digitalis and may lead to digitalis toxicity without change of dosage. Thus, it is important to check the serum potassium and, if necessary, supply oral potassium chloride.

9. Antibiotics. Many infants with heart failure have pulmonary overcirculation and are unduly susceptible to respiratory infections. Even after auscultation, X-ray, and white blood cell count, it is often difficult to be sure whether or not pulmonary infection is present. Flaring of the ala nasi, grunting, or intercostal retractions suggest the presence of pneumonitis and the need for antibiotic therapy.

10. Morphine sulfate. This drug is indicated only in the rare instance where a baby in heart failure is restless and agitated. One mg. per 10 lb. of body weight is given subcutaneously.

Emergency measures. If the condition of an infant with heart failure is deteriorating rapidly, more aggressive measures may be indicated particularly if the baby has an operable heart lesion.

1. Rotating tourniquets. "Medical venesection" by tourniquets placed around the upper arms and thighs can be a lifesaving measure. The major indication is pulmonary edema. Three tourniquets are applied tightly enough to obstruct venous but not arterial circulation, and one tourniquet is moved every ten minutes so that the circulation of each limb is unobstructed for ten out every forty minutes.

2. Sodium bicarbonate. Acidosis accompanying severe heart failure may be respiratory or metabolic. If plasma pH falls below 7.0, with associated bradycardia or arrhythmia, the situation may be temporarily improved by the rapid infusion of sodium bicarbonate, 4 to 6 mEq. per kilogram of body weight.

3. Epinephrine infusion. Infants with severe heart failure due to left-to-right shunts may improve temporarily following epinephrine infusion.[3] This sympathetic neurohormone is believed to act by direct inotropic effect and as a coronary vasodilator. It should be given by a constant infusion pump at 0.5 to 1.5 μg. per kilogram per minute, with continuous electrocardiographic monitoring and frequent blood pressure measurement. Since the infant appears to become epinephrine dependent, there has to be a foreseeable conclusion. Usually, this will be surgery, such as division of a large patent ductus arteriosus. Epinephrine infusion should be continued throughout the period of induction of anesthesia and until the underlying cause of heart failure has been corrected. It should then be discontinued slowly, with monitoring of blood pressure and an electrocardiogram.

CONCLUSIONS

Early recognition and aggressive treatment of heart failure in infants is mandatory if the mortality from structural congenital heart disease is to be further reduced.

Heart failure occurring in the neonate with a congenitally malformed heart usually indicates a rapidly deteriorating situation, and aggressive medical treatment should be started immediately. Hemodynamic and angiographic studies are often indicated to obtain a firm anatomic and physiologic diagnosis and undertake appropriate surgery.

In the case of the older infant presenting with heart failure, medical treatment usually will produce considerable improvement, although, in many instances, diagnostic studies are necessary for full appreciation of the pathophysiology.

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Electroencephalographic Responses to Sound and to Light in Premature and Full Term Neonates

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ALTHOUGH MATURATIONAL changes in the neonatal period are documented by the changing pattern of the electroencephalographic tracing, meaningful measurements of the rate of maturation are not obvious. In contrast, functional tests by sensory stimulation, using electroencephalographic methods, permit measurements of responsiveness at different age levels. In animal experiments primary evoked responses can be observed at the surface of the cortex, and changes in amplitude and latencies can be measured. In the human neonate, routine observations are limited to potentials conducted by scalp electrodes. In 1958 Ellingson[1] published his first paper on visual evoked responses in newborns.

Immediately after birth it is possible to register evoked potentials from the inion or from both occipital areas of the scalp over the visual cortex, when the retina is stimulated by light flashes. A definite maturational pattern has been established for response latencies of photically evoked potentials in the visual cortex. The latency is inversely proportional to the conceptional age of full-term and premature infants.[2] There are also secondary cortical responses to photic stimulation noted with longer latencies and also subcortical responses, which follow the stimulus after shorter intervals.

1. *Acoustic stimulation.* Primary cortical responses can also be obtained with acoustic stimulation in animal experiments.[3] In human beings the auditory cortex is buried in the Sylvian fissure and inaccessible to monitoring from the scalp. Present technique does not yield interpretable responses in the routine electroencephalogram using scalp electrodes but so-called secondary slow cortical responses can be summed up readily following acoustic or other sensory stimulation.[4,5] Auditory stimulation with modification of the signal produces different response amplitudes in the electroencephalogram (EEG).[6] The test is useful in audiometry, even though the response does not arise from the primary auditory cortical area (Davis).[7]

Slow cortical responses evoked by acoustic stimuli and described in older children and adults by P. and H. Davis can also be elicited in neonates.[8] The latencies, however, of the characteristic sequence of positive and negative waves are longer in the immediate newborn period.[9]

METHODS

The same children who were the subjects to photic stimulation, were also tested subsequently by acoustic stimulation. After considerable probing, the method of Hallowell Davis[4] was adapted for use in neonates in connection with an additive response computer of time-locked events (Mnemotron CAT) in order to demonstrate the secondary auditory responses in the electroencephalogram.

In older children and adults the most typical waves in response to auditory stimuli are a small vertex-positive wave with a peak (P_1) at 50 to 60 milliseconds (msec.) followed by a strong vertex-negative wave (N) at 95 to 105 msec. and then a large vertex-positive wave (P_2) at 170 to 200 msec. Davis believes that the overall pattern is composed of 3 or 4 components which may vary independently in amplitude, but which are fairly stable in latency. Williams, Tepas, and Morlock;[10] Weitzman and Kremen;[11] and McCandless and Best[12] confirmed the average latencies (P_1 50, N 100, P_2 175 msec.).

A noisemaker was constructed, which generates clicks in a loudspeaker. The electrical pulse

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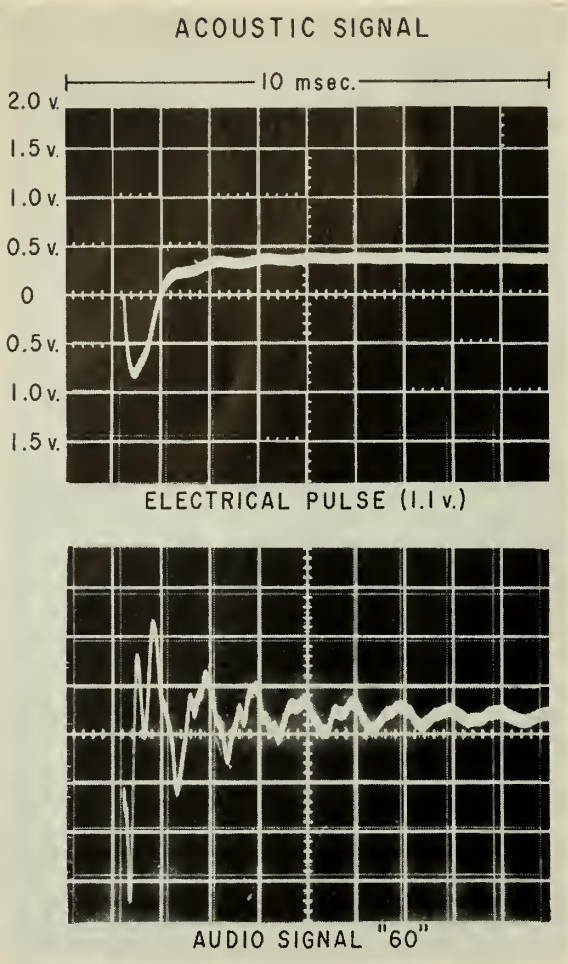


Fig. 1

lasts about 1 msec., the membrane of the loudspeaker swings about 10 msec. at the setting which was used in all tests (Fig. 1). Its intensity is about 80 decibels above adult waking threshold, and as a rule, the blinking responses or Moro reaction to the click were absent. For threshold determination the intensity of the click could be modified.

The infant was placed in a sound proof chamber and could be observed through a window. He was placed on his side, facing the observer. The EEG was monitored over the vertex by the computer with the other bipolar lead in the preauricular area of both sides. The stimulus was offered at irregular intervals, not less than 1 second apart. This was done by hand or by a programmed tape with 45 holes, punched at random. The intensity of the generating current was kept constant; the number of added stimuli, however, was varied according to the response amplitude in individual cases.

In rare cases auditory responses to single clicks could be identified on visual inspection on the EEG tracing (Fig. 2). Usually 30 to 50 or more random deliveries were administered, before the computed tracing was taken off from the oscilloscope after it had been copied on the X-Y plotter (Fig. 3). Readings were only accepted when confirmed on repeat testing. Arbitrarily, the time span triggered by the stimulus and fed into the Mnemotron Computer was set at 500 msec. Thus, later responses were not recorded.

Most infants were tested during natural sleep. The depth of sleep was assessed by clinical impression and confirmed by the characteristic EEG tracing. The level of alertness, however, could change during testing. In such cases the attached judgment: "awake," "drowsy," "light sleep," "deep sleep" refer to the domineering behavior. Deep sleep was confirmed by alternating tracing in the EEG whereas light sleep is characterized by rudimentary spindles. When the observer noted any facial or body movements, the switch to the Mnemotron Computer was turned off because movements throw the computer out of balance due to the magnitude of the artefacts.

RESULTS

In roughly two-thirds of 317 neonates tested according to the described method, a P_2 wave could be identified and found again on retesting. P_1 was obvious in less than one-third of the same cases. The negative trough (N_1) between both vertex positive waves occurred at the same latency interval on repeated examination in about 55% of this series.

Figure 4 shows the mean peak latencies ± 1 standard deviation for the 2 vertex positive waves and the N_1 wave plotted against conceptional age. Looking at the overall sample averaged across subjects between thirty-seven and forty-three weeks of conceptional age, no progressive shortening of response time can be detected with age in the immediate neonatal period. Table 1 shows the results in full-term neonates.

Individual differences are considerable and are expressed by the large standard deviation.

TABLE 1
THE SLOW CORTICAL AUDITORY RESPONSE IN FULL-TERM NEONATES
(Conceptional age 40 weeks according to obstetrician)

Sex	*Positive response (%)*	*Number of infants with measurable P_2 latency*	*Mean latency of P_2 (msec.)*	*1 S.D.*
Boys	59	73	243	± 30 msec.
Girls	68	88	240	± 30 msec.

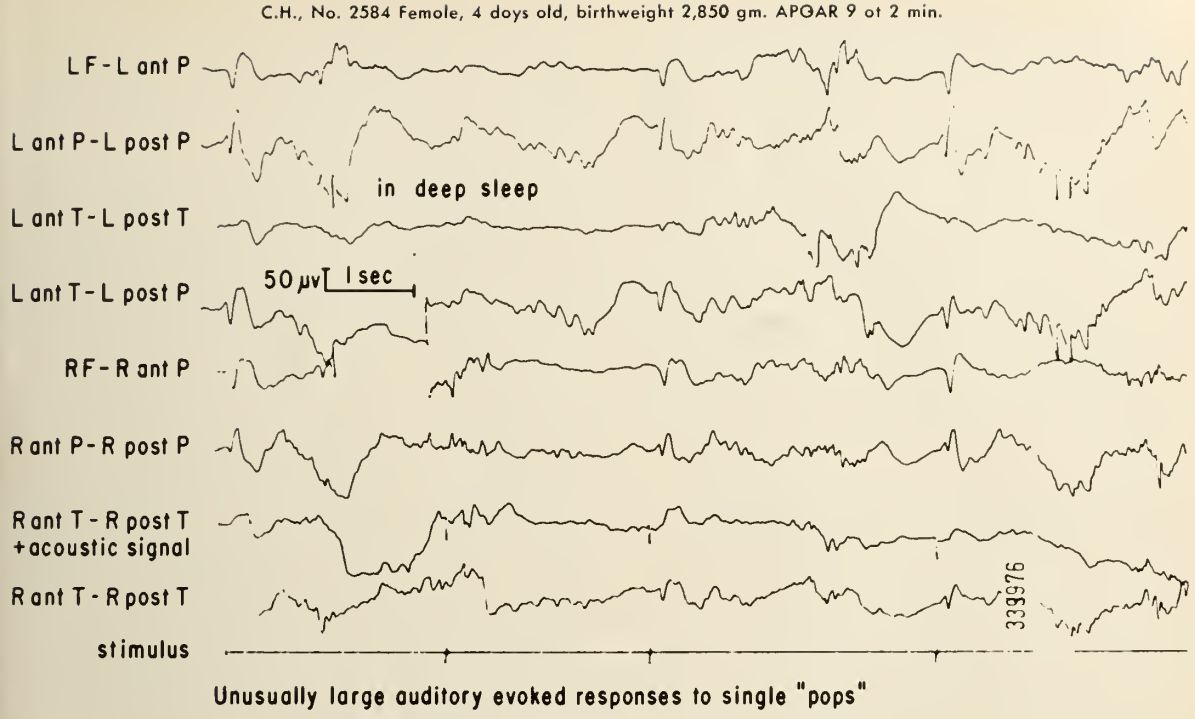


Fig. 2. EEG, acoustic stimulus, and evoked response

This does not prevent the successful application of the vertex potential in "objective" audiometry in children (H. Davis).[7] The difference between N_1 and P_2 is most easily identified and permits the measurement of amplitude, which is important in threshold determinations.

The best reproducibility of secondary auditory responses was obtained during sleep. The latency periods were slightly longer during sleep as compared to the waking state.

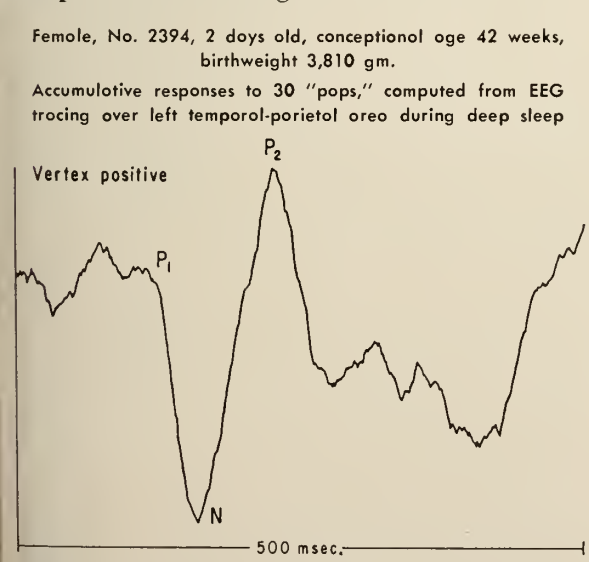


Fig. 3. Sequence of acoustic responses during the first 500 msec. after stimulation

On several occasions we had the opportunity to test hydranencephalic children. These children without much supratentorial cortex did not show the slow response to acoustic stimuli nor

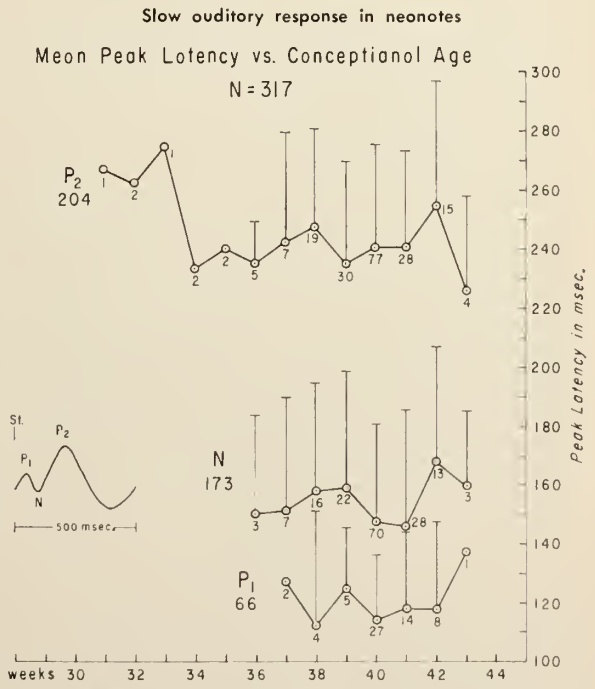


Fig. 4. Mean ± 1 standard deviation of slow cortical responses to clicks in neonatal period

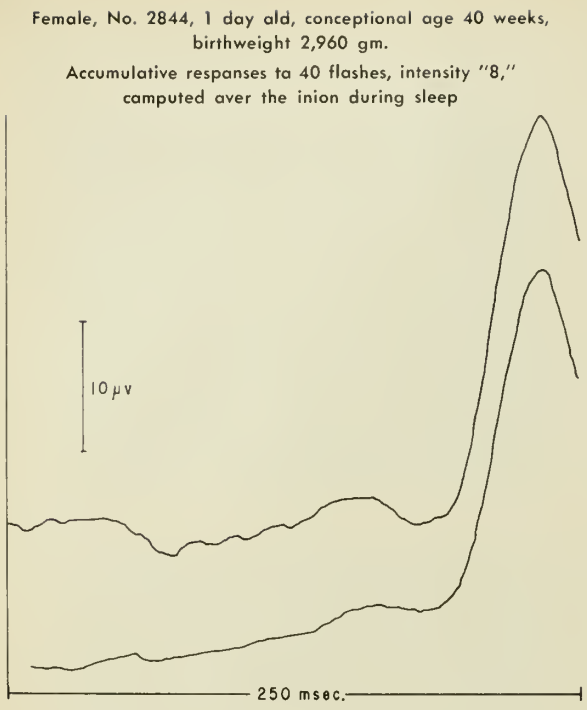


Fig. 5. Primary photic response over the inion

any brain wave activity. They did demonstrate muscular artefacts and, upon photic or acoustic stimulation, they showed a jerk or a blink and a complex waveform due to muscular artefacts. The cranial nerves were found to be intact and the reflex mechanism functioning. The characteristic slow cortical responses of H. Davis, however, remained absent in the computed tracing of the electroencephalogram.

2. *Photic stimulation.* The practical application of electroencephalographic auditory tests lies in threshold determinations.

Photic stimulation not only causes secondary responses in the vertex electroencephalogram but also primary responses can be measured with scalp electrodes over the inion and over the visual cortex. In premature infants, the primary responses in the occipital area appear with significantly longer latency than seen in normal, full-term neonates (Ellingson, 1958).[1] The conduction time permits the assessment of maturity at birth. Photic response activity and latency measurements vary according to conceptional age.[13]

METHODS

At the beginning of the present study a transportable, 8 channel Offner EEG apparatus and the standard Grass stroboscope were at our disposal. The stimulus consisted of single flashes, repeated at irregular intervals of not less than 1 sec. The tentative starting intensity of the standard Grass stroboscope was "8." The light intensity was diminished when the infant blinked or increased when no evoked response was noted (Engel, 1961).[14]

The conduction time from the onset of the stimulus to the EEG response is the latency period. Ellingson (1958)[1] chose as endpoint the peak of the response potential. This, however, is not always obvious because muscular potentials connected with blinks may obscure the maximal deviation of the potential.

Depending on visual inspection alone, latency was defined as the time from the onset of the stimulus to the first major deviation from the baseline. This was possible in 75% of our cases, full-term and premature infants. With the use of the Mnemotron Computer of Average Transients, it now is possible to register responses to appropriate photic stimulation in all cases with functional visual cortex (Fig. 5).

In full-term infants, we computed over a span of 250 msec. triggered by the stimulus impulse and measured to the first distinct wave. Only rarely did the time span have to be extended in immature infants in order to demonstrate the onset of the major waves. As a rule, the electroencephalographic testing was done on the second or third day of life and sometimes only hours after birth. Premature infants were retested several times, until they reached a weight of 2,500 gm. and were discharged from the hospital.

The conceptional age was calculated according to the mother's menstrual calendar. If this did not fit with the infant's appearance and body measurements, the obstetrician's estimate was used, usually based on weight. In many cases, however, the birth weight is misleading. Among twins and triplets there are many premature births. Here, the pediatric evaluation by bone age, crown-rump length, and clinical impression also was included in our best estimate of conceptional age at time of the EEG.

RESULTS

In a previous series of 1,700 neonates we have tested the photic responses in premature, full-term, and postmature infants and found the latency measurement to be inversely proportional to the conceptional age at the time of the EEG (Engel and Butler,[2] 1963; Engel,[13] 1965).

Figure 6 is a scattergram with the regression line computed from the same population of neonates. It is assumed that a straight line adequately characterizes the relationship between

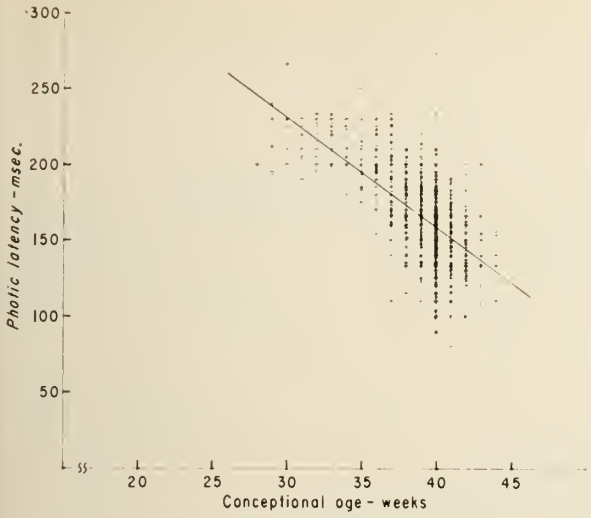


Fig. 6. Regression line and scattergram relating photic latency to conceptional age

photic latency and the conceptional age of the restricted range between 32 and 43 weeks of conceptional age.

In a recent study limited to 341 single infants at term observed with the help of the adding computer the following means and standard deviations were obtained for photic latency (PL) and photic peak latency (PPL):

TABLE 2

PRIMARY PHOTIC RESPONSES IN FULL-TERM NEONATES

1) MEAN conceptional age at time of EEG according to last menstrual period	283.0 ± 14.6 days
2) MEAN weight at time of EEG	3,180.8 ± 516.9 gm.
3) MEAN PL	157.4 ± 20.2 msec.
4) MEAN PPL	197.3 ± 24.5 msec.

Differences between the first few days of life are minimal (Ellingson, 1958).[1] Figure 7 confirms this statement in a transverse observation by plotting means ± 1 standard deviation of PL against age in days after birth. The graph also demonstrates that girls seem to react faster than boys at term. This observation was confirmed with statistical significance ($p < .001$) whenever the sample was large enough and it was seen in 3 different races we tested: Negro, Caucasian, and Oriental (Engel, Crowell, Nishijima).[15]

In individuals the standard deviation is small, when tests are made on an hourly basis for a twenty-four-hour period. Hourly measurements of 50 added evoked responses during a period of twenty-five hours in a normal girl, 4 days old, whose weight was 3,380 gm., and showed a mean PL 155.6 ± 4.3 msec. and a PPL 193.4 ± 7.7 msec., regardless of wakefulness, drowsy states, or sleep.

Sex and race can explain only part of the differences between individuals.[15] Another part remains still unexplained, but a major contribution is made by the neurologic maturity at the time of birth. If a human fetus were retarded in maturation as well as in growth, the newborn would have the physical characteristics as well as the size of an immature infant in spite of a perhaps normal length of gestation. In fact, many prematurely born infants failed to show the shorter response latency, found in full-term infants.

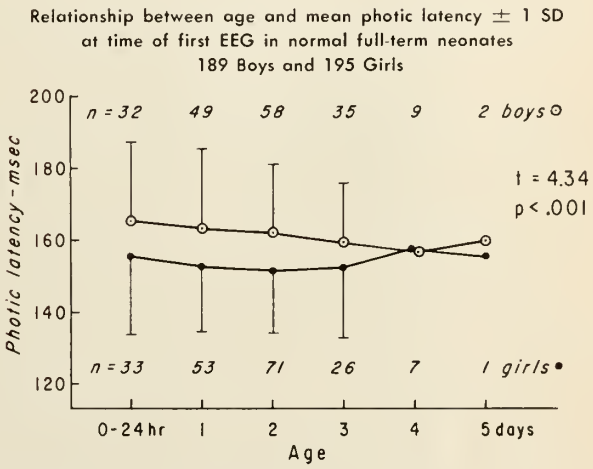


Fig. 7. Sexual differences during first few days after birth

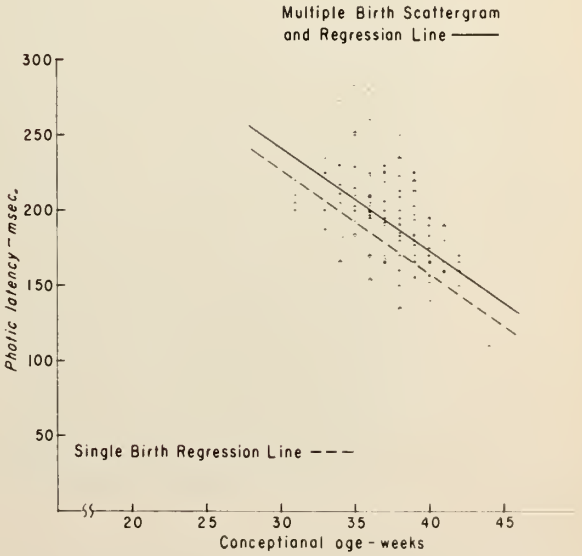


Fig. 8. Regression line and scattergram of multiple births compared with regression line of single births relating photic latency to conceptional age

when the former reached the conceptional age of 39 to 40 weeks (Engel, 1965).[13]

A similar observation can be made if a statistical comparison of multiple versus single birth is undertaken. Among the multiple births are many prematures. The regression line based on our observations on multiple births has the same slope as the computation from single birth observations, but it is elevated (Fig. 8). This indicates that the rate of change is equal, but the average photic latency at corresponding conceptional ages is longer in the multiple birth group. The majority of twins or triplets has not quite reached the fast responsiveness of single individuals at term. They seem to mature with a slight delay. This is in agreement with many clinical observations on the maturational rate of premature infants.

SUMMARY

1. The slow cortical responses evoked by acoustic stimuli and described by H. Davis in older children and adults can also be measured in the vertex potentials of newborn electroencephalograms with a longer latency period.

2. The most easily measured wave in the series of secondary responses is the vertex positive wave with a mean peak latency (P_2) of 241 $\pm$ 30 msec. in contrast to older children and adults ($P_2 = 170$-200 msec.).

3. Photic stimulation permits the measurement of primary evoked responses in the electroencephalogram over the inion. The mean photic latency (PL) is 157 $\pm$ 20 msec. and the peak latency (PPL) = 197 $\pm$ 24 msec. in single birth full-term infants.

4. Photic latency is inversely proportional to conceptional age in the neonatal period and its measurement adds an independent estimate of gestation to other criteria of maturation.

5. Sex explains only part of the great individual differences in neonatal electroencephalograms. Girls, as a rule, seem to be more advanced than boys at the time of birth.

6. Not all premature infants reach a state of equal maturation with normal full-term, single birth infants when the EEG is repeated at the conceptional age of 40 weeks.

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The author wishes to express appreciation to Miss Ruth Burnett, R.N., and Mrs. Mary Sorenson, both electroencephalographic technicians, for skillful technical assistance and to Donald R. Jensen, assistant professor of statistics, Virginia Polytechnic Institute, for statistical analysis of data.

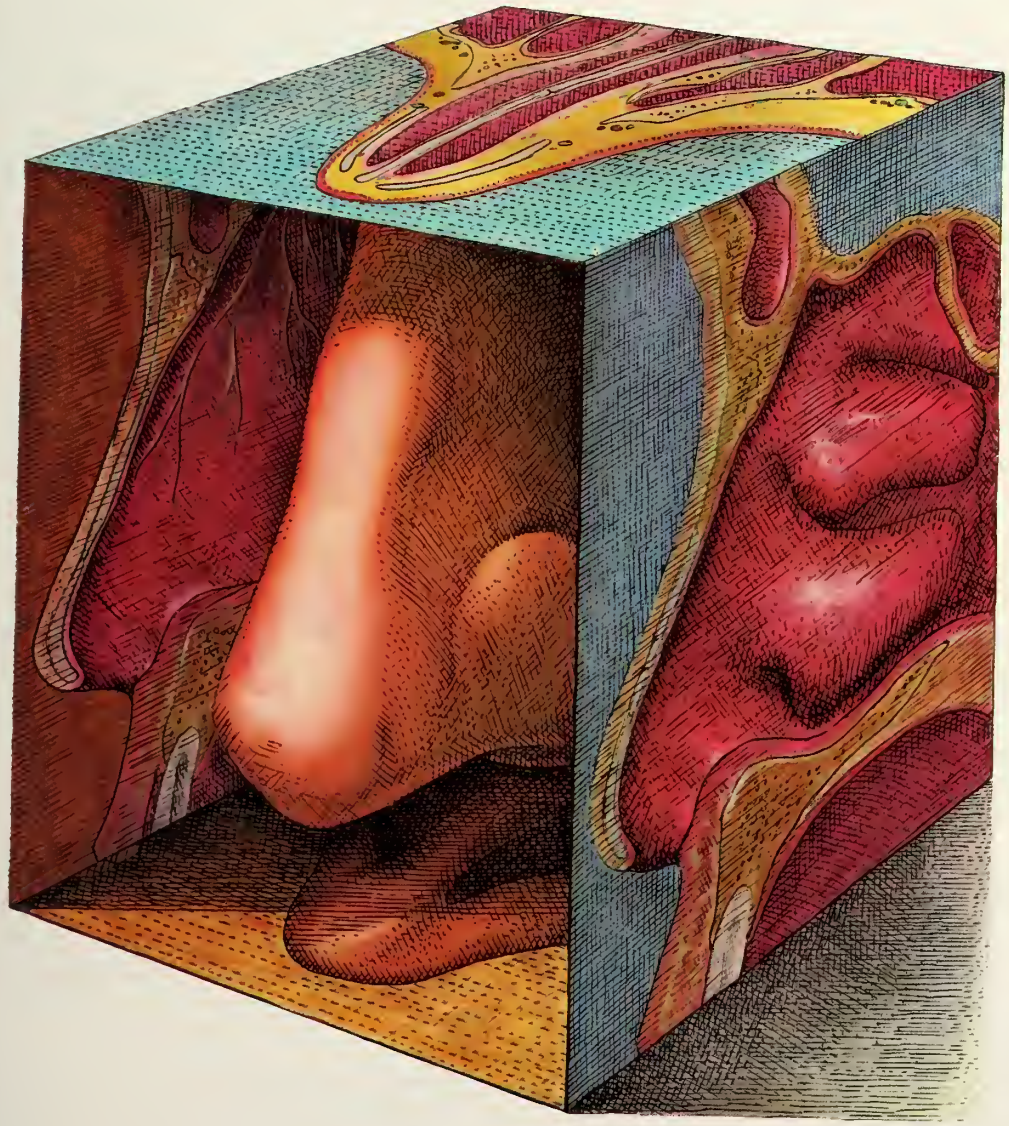
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DORSEY Season

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this issue: the ubiquitous world of summer allergies



the ubiquitous world of summer allergies

Donald L. Unger, M.D. • Clinical Assistant Professor, Department of Medicine (Allergy), Stritch School of Medicine (Loyola).

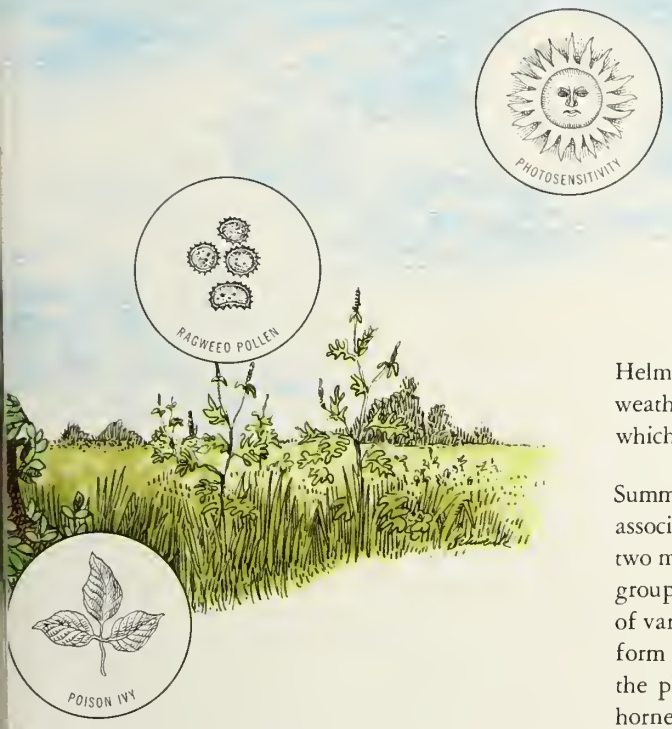
In the Spring a young man's fancy lightly turns to thoughts of—allergies. This is at least true of the 10% of the population who have hay fever and the 4% who have asthma.[1] The snow melts, the trees blossom and the noses run. Patients who were fine all winter may not be enthralled by the sight of the first robin or the blossoming of a crocus, for their appearances may precede the "sneezin' season."

Allergies in general can be divided into winter allergies and summer allergies. In the winter the main problems are inside the house: e.g. dogs, cats, dust and feathers. Houses in the northern half of the country become so dry that it becomes essential to add humidity to the home; this is a far cry from the damp summer months with the moldy basements and need for dehumidifiers.

Early in April trees begin to pollinate, with each tree having about a two week period of pollination. A particular patient may be sensitive to only one tree and thus have his hay fever for such a short time that he thinks he has a cold.[2] The entire tree season starts about April 1 and ends about Memorial Day, although all hay fever seasons are blurred and prolonged in the southern part of the country. Tree pollen is usually very heavy and a person may well have most of his exposure from those trees immediately surrounding his home.

Grasses pollinate from about May 15 until July 4, and cause "rose fever." Grass pollens are somewhat lighter and more buoyant than tree pollens, and are much more ubiquitous. While there are several varieties of grasses in the United States, they are so closely related antigenically that a person sensitive to one is generally sensitive to them all.[3] Thus, while the tree season is really several small seasons intertwined, the grass season will usually result in symptoms for a more prolonged period. Obviously, a grass-sensitive patient will have trouble only when grass is pollinating—he will have to think of another excuse not to mow the lawn after July 4.

Ragweed is the "Big Daddy" of them all in the eastern two-thirds of the country. Pollination is generally from mid-August until the end of September, with the predicted lower counts and longer seasons



in the southern part of the country. Ragweed is a very light pollen which may be windborne for hundreds of miles. An interesting study was made in New York City, in which 90% or more of the ragweed plants were destroyed in three of the five boroughs; pollen counts done during the season were virtually identical in all five.[4]

Ragweed is, of course, the most common cause of hay fever and is associated with an incredible loss of man hours from work each year. Many is the patient who travels to areas where the pollen count is low, just to avoid having symptoms. There is no ragweed anywhere in the world except the United States and portions of Canada and Mexico.

While molds are present through the year, the most important ones predominate from April until November. An old wives' tale has ragweed ending with the first frost, when actually it ends a good month earlier. It is Alternaria—the kingpin of the molds—that meets a sudden demise with the first frost. Alternaria-sensitive patients are in their glory when there is snow on the ground, and might be ideally suited to man the radar stations in Alaska. In September and October, Alternaria counts are at their highest, perhaps associated with the burning of leaves. Other molds such as Hormodendrum and Helminthosporium are associated with the warmer weather, as opposed to Penicillium and Aspergillus which are household molds.

Summer also means the return of our much maligned associates—bugs. Insects cause allergic symptoms by two methods: the bite or sting of the Hymenoptera group, and the inhalation of particles of the bodies of various insects. Wasp stings are the oldest known form of allergy, as they caused the death of one of the pharaohs in ancient Egypt.[5] Bees, wasps and hornets account for many deaths in this country, and those sensitive to them should carry special treatment kits at all times; a few minutes delay in the administration of epinephrine to such a patient, might be the difference between life and death. Inhalation of particles of insects may cause sneezing and wheezing in a susceptible individual.[6] Both of these forms of insect allergy may be benefitted by hyposensitization.

The insect recognizes no professional bounds. He is as apt to bite the physician as the patient. So this season, beware of bugs. And beware, too, of poison ivy. That pleasant stroll through the woods and underbrush with the Boy Scouts might turn into a

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nightmare for the botanically uninitiated in the causes of rhus dermatitis (poison ivy, poison oak and poison sumac). Although you may have been careful, your dog may not have noted that it wasn't clover he jumped through, but poison ivy. His return to your side may give you the rhus dermatitis that you so carefully avoided. That heavenly campfire may be emitting particles of rhus oil to produce an airborne contact dermatitis of the exposed areas of the body.

Another fascinating, but rather infrequent type of summer allergy is physical allergy. Some people sneeze on exposure to sunlight, while others break out in rashes, usually on the exposed parts of the body. These rashes may well follow the administration of various photosensitizing drugs, e.g. demethylchlortetracycline.[7] Another form of physical allergy and one that may be lethal in the summer, is cold allergy. Yes, I mean cold allergy, not heat allergy. The cool dip on a hot day with its consequent sudden chilling of the body, may be the coup de grace for a cold sensitive patient.[8] It is customary to write "heart attack" on the death certificate, even though the victim may have been an 18-year-old boy who looks like a Greek god.

Lest the reader be depressed by this saga of afflictions associated with the warmer months, perhaps he should remember that it is also a time for swimming, baseball, lying in the sun and taking that long-planned vacation. So let's all join in a chorus of "In the Good Old Summertime," as we sneeze, wheeze and scratch. Be careful of your suntan lotion, however; it may cause you a contact dermatitis.

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Book Reviews . . .

Ocular Therapy: Complications and Management

edited by IRVING H. LEOPOLD, M.D., *157 pp., ill., C. V. Mosby Co., St. Louis, 1966. $11*

This book is primarily a collection of articles published in conjunction with two recent eye symposia. Readers will recognize contributing authors as some of this country's leading ophthalmologists in the field of ocular therapeutics and pharmacology. The text is geared for the practicing ophthalmologist, who can rapidly assimilate considerable practical information in one or two evenings of reading. The articles are printed in large clear type with generous spacing and most chapters have an extensive bibliography.

By no means is this text exhaustive and inevitably, omissions and oversimplifications occur, as in any book of this type. The result is a staccato presentation of some highlights and the quality of each chapter is variable. I am forced to conclude that the contributions of some authors were included to enhance the prestige of this text. The articles by Drs. Frank Newell and Bernard Becker are interesting from an experimental point of view, but are only three pages in length and of no clinical value. Only a brief section is devoted to autonomic drugs in ophthalmology, although these are probably the most commonly used drugs in this field.

The most detailed section is that on anesthetic drugs in ophthalmology. This section makes an excellent reference because it is not overly detailed, yet is sufficiently comprehensive for most practitioners. The chapters on the hazards and side effects of the newer antibiotics, cardiovascular, and renal agents are current and informative. Other chapters that are enlightening are those on ophthalmic agents in pediatrics, tranquilizing agents, and nonsteroidal antiinflammatory agents. The sections devoted to virus chemotherapy and topical corticosteroids are interesting, but experimental in nature, and did not fit in with the general clinical tenor of the book.

SIDNEY NERENBERG, M.D.
Minneapolis

Pulmonary Embolism

by FRANK GRAY, JR., M.D., *234 pp., ill., Lea & Febiger, Philadelphia, 1966. $8.50*

The book is divided into nine chapters with the major chapters being: "The Embolus," "Pathologic Effects of Embolism," "Pulmonary Infarction," "Physiology of Pulmonary Embolism," "Clinical Diagnosis," and "Management of the Patient with Embolism."

In general, the chapters were well written and succinct and provided considerable amounts of useful information. The chapter entitled "The Embolus," however, could be singled out for excessive verbosity. The material is in more extensive detail than most readers would find practical.

The book, in general, provides a detailed and well documented discussion of virtually all aspects of pulmonary embolism. The section on case histories is quite useful since it provides illustration of the diverse and subtle modes by which embolism may present.

This book would provide valuable reading for virtually any physician since pulmonary embolism appears to be a risk involved in the care of patients in practically all specialties in medicine. It is somewhat more extensive in detail than many clinicians would find helpful for routine reading. It would, however, be quite useful as a reference work.

ROBERT E. OLSON, M.D.
Minneapolis

Cellular Concepts of Rheumatoid Arthritis: Holbrook Memorial Symposium

edited by C. A. L. STEPHENS, JR., M.D., *and* A. B. STANFIELD, M.S., *212 pp., ill., Charles C Thomas, Springfield, 1966. $14.75*

This volume contains the individual lectures and ensuing discussions from the 1964 Holbrook Memorial Symposium held on the campus of the University of Arizona.

Because the concepts presented are common to connective tissue in general, with only possible relevance to rheumatoid arthritis, the title is misleading. Several chapters stand out, however, particularly a discussion of cellular morphology of the fibroblast and related forms by Keith Porter. The electron microscopic studies are beautiful and complement the discussion well. Cellular function is demonstrated, including a secretory apparatus active in producing collagen and mucopolysaccharides as well as the important endoplasmic reticulum. A basic knowledge of morphology is essential to understanding the flood of current literature on cellular morphology.

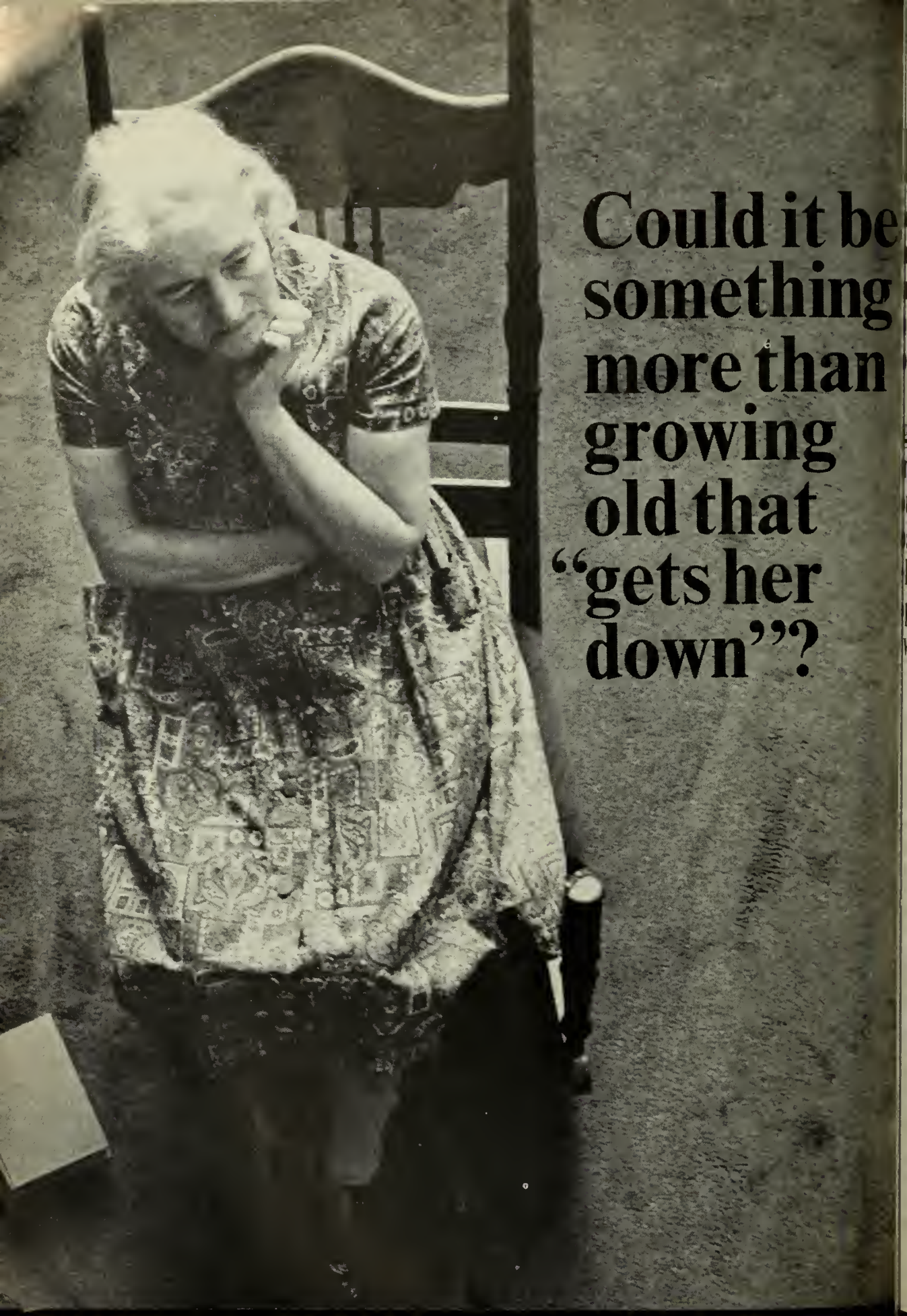
Techniques of cell culture and experimental production of homologous disease are the subjects of other chapters. The discussion of time-lapse movies would be more meaningful if one were attending the meeting.

Dr. William Dameshek presents his well-known ideas on auto-immunity and describes them as "mildly speculative." This speculation is affirmed by the University of Oregon's Edwin Osgood, who disagrees with the concept and labels his own explanation "cross-immunity." Even Dr. Dameshek's immunoblast comes under fire, being called merely a lymphocyte in a particular phase of cell division. Dr. Dameshek's objection to the semantics involved brings forth spirited illumination on both sides.

Dr. Eugene Barnett's lecture on antinuclear factors is a competent discussion of a clinically confusing subject. The discussions following each chapter are interesting and pertinent. Verbatim, unedited discussions such as these, however, have a tendency to fall flat with reading.

The field is changing rapidly, and what was true in 1964 may not stand up well in 1967. One wonders why there was such a long delay in publication. Subjects in current vogue, such as lysosomes, different synovial cell types, and possible infectious agents, are mentioned briefly. The book belongs in reference libraries but will appeal to all interested in learning basic methods of arthritis investigation. Dr. Dameshek's ideas on autoimmunity, with the interesting rebuttals, and the cellular micrographs will appeal to most physicians.

GERALD T. MULLIN, JR., M.D.
Minneapolis



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*Multiple of adult Minimum Daily Requirement supplied.
†The need for these substances in human nutrition has not been established.

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News Briefs . . .

North Dakota

DR. W. GENE SCHROEDER of the Grand Forks Air Base has opened a part-time office in the Nelson County Memorial Hospital.

* * * *

DR. E. H. GOODMAN of Napoleon has moved his office into the new clinic building.

* * * *

DR. CARL J. BAUMGARTNER was reelected president of Bismarck Clinic, a corporation owning the medical properties of Quain & Ramstad Clinic.

* * * *

DR. DENNIS E. WOLF has joined the staff of the Rodgers and Gumper Clinic of Dickinson.

* * * *

DR. LOUIS B. SILVERMAN of Grand Forks has been named medical director of an evaluation center for exceptional children to be established at the State Medical Center at the University of North Dakota. The project is the result of a grant of $97,000 per year for a three-year period from the State Department of Health.

* * * *

DR. HUGH W. HAWN of Fargo was presented a U.S. Department of Health, Education, and Welfare award for helping raise the health of the Indian population of the White Earth Reservation.

* * * *

DR. NEVILLE JONES, formerly of London, England, has joined the staff of the Hebron Medical Clinic.

Minnesota

REGENTS' PROFESSORSHIPS of the University of Minnesota were bestowed upon DR. WESLEY W. SPINK, professor of medicine, and DR. MAURICE B. VISSCHER, professor of physiology. The honor is the highest the university can confer upon its most distinguished professors and carries with it a $5,000 annual gift for as long as the faculty member remains on the university staff.

* * * *

DR. T. G. MARTENS, consultant in ophthalmology in the Mayo Clinic, Rochester, has been elected chairman of the board of directors of Preschool Survey of Vision and Hearing, a public-service organization sponsored by the Minnesota State Medical Association.

* * * *

DR. NORMA SCHNABEL has joined the medical staff of the Northwestern Clinic in Crookston.

* * * *

DR. GEORGE M. MIKS, formerly of Eveleth, has joined the staff of East Range Clinics, Ltd., in Aurora.

* * * *

DR. DONN G. MOSSER of Minneapolis has been reappointed to the American Medical Association committee on medicine and religion.

DR. BAYARD FRENCH has taken office as president of the Hibbing General Hospital medical staff.

* * * *

REAPPOINTED to posts in the American Medical Association were the following Mayo Clinic consultants: DR. GEORGE LOGAN as principal representative on the interspecialty committee of the American Academy of Pediatrics; DR. J. H. TILLISCH to the council on occupational health; DR. JAMES CAIN to the council on national security; and DR. J. R. BROWN as principal representative on the interspecialty committee of the American Academy of Neurology.

* * * *

DR. S. C. G. OELJEN has been elected president of the Waseca County Medical Society.

* * * *

DR. S. S. GISLASON of St. Peter has returned from a three-week tour of outstanding universities in Europe.

* * * *

DR. ROBERT G. CARTER, formerly of Mora, has taken a residency in radiology at Swedish Hospital in Minneapolis.

* * * *

DR. M. MELVIN GOLDFINE of Oakland, Calif., a graduate of the University of Minnesota, has been elected to the board of trustees of the Minnesota Medical Foundation.

* * * *

DR. HERBERT L. HUFFINGTON of Waterville has been appointed to the University of Minnesota Board of Regents.

* * * *

DR. HARRY B. HALL, a clinical professor of orthopedic surgery at the University of Minnesota, has been named by Gov. Harold LeVander to head a committee to study Minnesota mental hospitals. Other physician members of the committee are: DR. RALPH ROSSEN, director of the L. E. Phillips Psychobiological Research Laboratory of Mount Sinai Hospital, Minneapolis; DR. MAGNUS PETERSON, Rochester psychiatrist; DR. JOHN HAAVIK, staff psychiatrist of the Duluth Mental Hygiene Clinic; DR. CHARLES HABERLE, staff psychiatrist of the Minneapolis Clinic of Psychiatry and Neurology; DR. HOWARD ROME, chief consultant of the section of psychiatry of the Mayo Clinic, Rochester; and DR. JOHN ANDERSON, chairman of the pediatrics department of the University of Minnesota Hospitals.

* * * *

DR. MARK E. ODLAND of Detroit Lakes has been named president-elect of the Heart of the Lakes Chapter of the Minnesota Academy of General Practice.

* * * *

DR. H. P. BARTHOLDT of Cook attended a three-week international medical meeting in March in Zurich, Switzerland.

* * * *

DR. J. B. SANFORD, Duluth surgeon, spoke recently at an American Cancer Society forum in Floodwood.

* * * *

DR. C. GORDON WATSON was named chief of staff at Fairview-Southdale Hospital in Minneapolis.

* * * *

DR. PETER DENT, an instructor in the University of Minnesota School of Medicine, has been awarded a five-year, $75,000 research grant by the Leukemia Society of America.

(Continued on page 22A)

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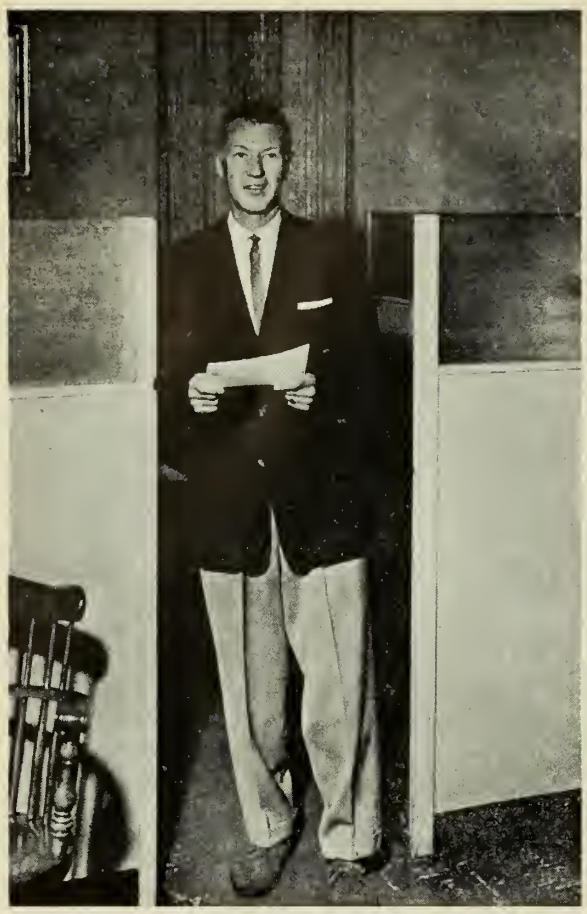
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(Continued from page 21A)

DR. JULES FEINSTEIN was elected president of the medical staff of Mount Sinai Hospital in Minneapolis.

* * * *

DR. JOHN B. RUTLEDGE of Detroit Lakes was presented a U.S. Department of Health, Education, and Welfare award for helping raise the health of the Indian population of the White Earth Reservation.

South Dakota

DR. JAMES D. COLLINS, a native of Trail City, will establish a medical practice in Hoven this summer.

* * * *

CONFIRMED officially in the capacities in which they have been serving for some time are DR. T. V. NIEMANN, chief of the medical service, and DR. W. L. MEYER, chief of the domiciliary medical service, of the Veterans Administration Center, Hot Springs.

Deaths . . .

DR. EARL BELL WEIBLE, 86, Barnesville, Minn., died February 8. A graduate of Baltimore Medical College, he practiced at Berthold and Abercrombie, N.D., for many years.

* * * *

DR. WALTER S. BROKER, 73, Battle Lake, Minn., died February 25. He began his general practice in Battle Lake and later served as superintendent at the Otter Tail County and Fair Oaks (Wadena) sanitoriums.

* * * *

DR. ROBERT L. BOWEN, 79, pioneer Hibbing, Minn., surgeon, died February 26. He was a founder of Hibbing's Mesaba Clinic in 1940 and played a major role in organizing the Hibbing General Hospital.

* * * *

DR. KATHERINE A. NYE, 80, St. Paul, Minn., died March 2. She was the only woman graduate in her 1914 University of Minnesota medical school class and was in private practice in St. Paul from 1915 to 1965.

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GENERAL PRACTITIONER wonted—to join established practitioner. Located 65 miles north of Minneopolis-St. Poul oreo. Populotian over 3,000. Hospital in tawn. Good schools, churches, ond fine fishing and recreotianal area. Contact A. C. Peterson, M.D., 200 E. Forest, Mora, Minnesoto 55051.

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1 Shermon, L. F.: Minnesota Med. 48.605. May, 1965. 2 Sherman, L. F. Personal Communication.

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CORTENEMA consists of a suspension of partially solubilized hydrocortisone alcohol in an aqueous vehicle. Thus, it provides the potent anti-inflammatory effect characteristic of the corticosteroids. Because hydrocortisone alcohol is absorbed to some extent from the colon, CORTENEMA provides both topical and systemic actions.

Indications: CORTENEMA Hydrocortisone Retention Enema is indicated as an adjunct in the treatment of idiopathic nonspecific ulcerative colitis. It is most useful in cases involving the rectum, sigmoid and left colon, where it exerts a topical effect. Due to its systemic action, CORTENEMA has proved effective in certain cases extending to the transverse and ascending colons.

Rectal steroid therapy brings about prompt symptomatic and proctologic remissions in a significant proportion of patients, but it apparently does not alter the recurrent and progressive nature of ulcerative colitis. Therefore, this form of treatment is not recommended for prophylaxis during periods of remission.

Contraindications: Local contraindications to the use of intrarectal steroids include obstruction, abscess, perforation, peritonitis, fresh intestinal anastomoses, extensive fistulas and sinus tracts. Tuberculosis (active, latent or questionably healed), ocular herpes simplex, and acute psychosis are usually considered absolute contraindications to the use of corticosteroids.

Relative contraindications include active peptic ulcer, acute glomerulonephritis, myasthenia gravis, osteoporosis, diverticulitis, thrombophlebitis, psychic disturbances, pregnancy, diabetes, hyperthyroidism, acute coronary disease, hypertension, limited cardiac reserve, and local or systemic infections, including fungal or exanthematous diseases. Where these conditions exist, the expected benefits from CORTENEMA must be weighed against the risks involved in its use.

If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

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References: 1. Musser, Ruth D., and Shubkagel, Betty L.: Pharmacology and Therapeutics, ed. 3, New York, Macmillan Company, 1965, p. 363. **2.** Council on Drugs, American Medical Association: New Drugs 1965, Chicago, American Medical Association, 1965, p. 157. **3.** Modell, Walter (Ed.): Drugs in Current Use 1966, New York, Springer Publishing Company, Inc., 1966, p. 77.

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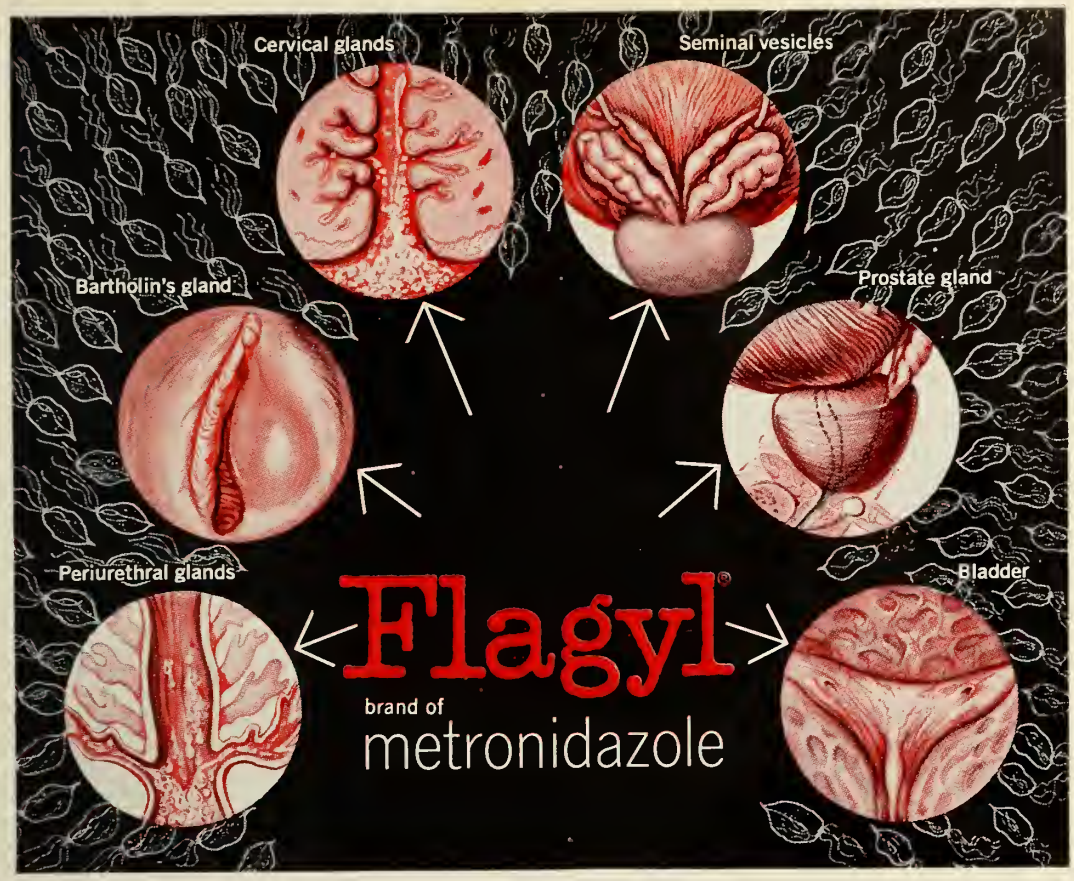
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CONTENTS

SYMPOSIUM—
Physical Medicine and Rehabilitation—PART II

Problems of the Bladder and Bowel in Patients with Myelopathies 187
Ann H. Schutt, M.D., Thomas L. Pool, M.D., and Gordon M. Martin, M.D.

Perceptual Problems of the Hemiplegic Patient 193
Robert R. Sawtell, M.D., and Gordon M. Martin, M.D.

Physiology of Balance 197
Frederic J. Kottke, M.D.

History of Physical Medicine at the University of Minnesota from 1945 to 1965 203
Mildred E. Olson, B.S., M.T. (A.S.C.P.)

The Relationship of the Staff Physician to the Department of Physical Medicine and Rehabilitation of a Community Hospital 207
Miland E. Knapp, M.D.

Two-Chambered Right Ventricle Associated with Primary Pulmonary Hypertension 210
A. F. Golwalla, M.D., S. D. Store, M.D., S. G. Kinare, M.D., and V. N. Acharya, M.B.B.S.

Therapy of Hemangiopericytoma with Pulmonary Metastasis 213
William Bogedain, M.D., and John Carpathios, M.D.

Owen H. Wangensteen 216
J. Arthur Myers, M.D.

Book Reviews 11A

News Briefs 18A

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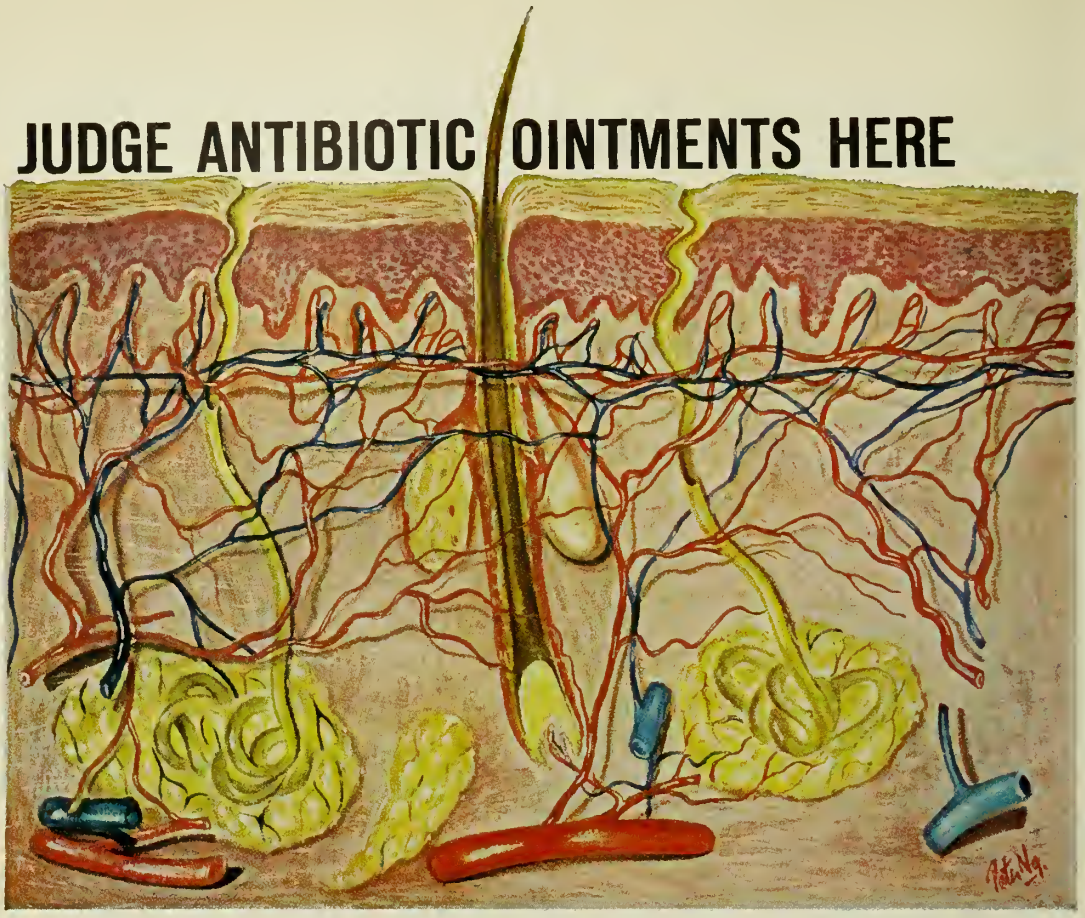
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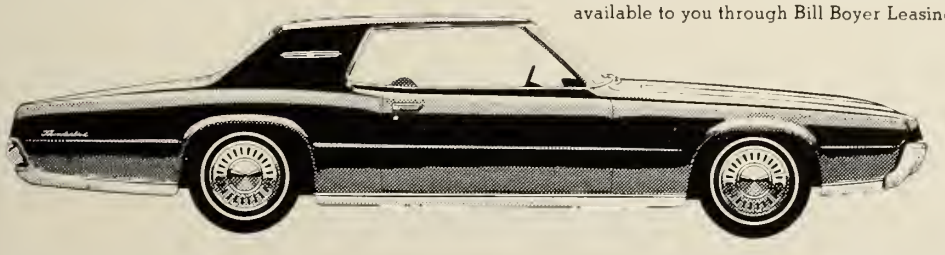
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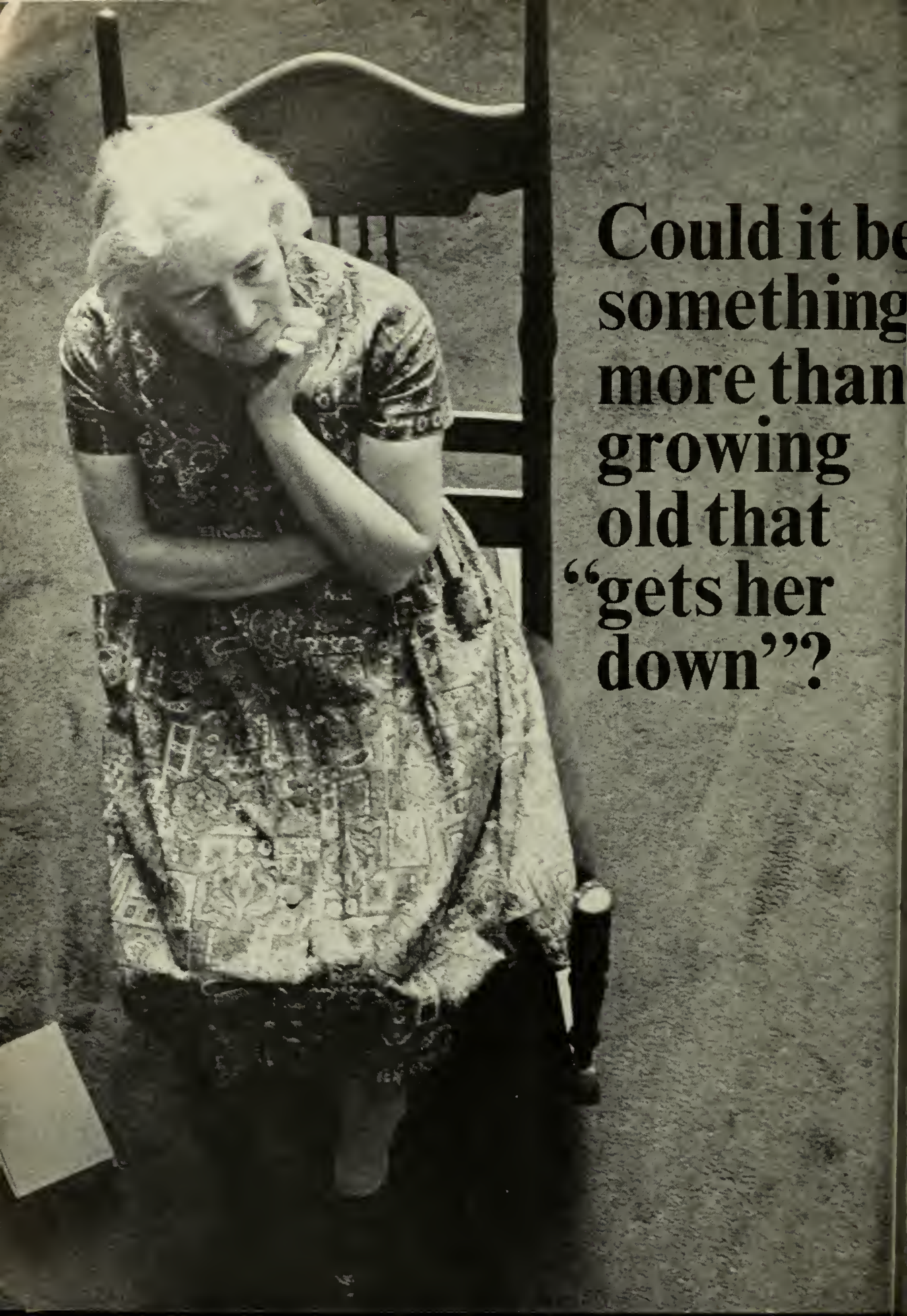
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SYMPOSIUM — Physical Medicine and Rehabilitation — PART II

Problems of the Bladder and Bowel in Patients with Myelopathies

ANN H. SCHUTT, M.D., THOMAS L. POOL, M.D., and GORDON M. MARTIN, M.D.

Rochester, Minnesota

Disorders of function of the bowel and bladder are only two of the many problems of patients with myelopathies. An understanding of the normal neurophysiology of the bladder and bowel is helpful to those who have to deal with functional disturbances caused by lesions situated at different levels of the spinal cord. Each treatment and rehabilitation center has its own slightly different routine for the management of disorders of the bowel and bladder, although, in general, at most centers the concepts of such management are essentially similar. A general plan for the care of the bladder and bowel among these patients is suggested, although opinions vary slightly concerning the specific care of the individual patient. Some of the more frequent problems and complications of dysfunction of the bowel and bladder among patients with myelopathies require immediate and special treatment.

NEUROPHYSIOLOGIC ASPECTS OF A NORMAL BLADDER

Vesical dysfunction is not specific to neurologic disease but is related to the extent, location, and duration of the neurologic lesion involving one or more of the pathways associated with the mechanisms of the bladder.[1] The bladder is innervated by parasympathetic, sympathetic, and somatic nerves.[2]

Urination involves a basic reflex act, partly under voluntary control. Urine is moved by peristaltic waves down the ureter and enters the bladder under pressure.[3] Backflow is prevented by valvelike action of the ureterovesical junction. Escape of urine from the bladder is prevented by constant contraction of both urethral sphincters. As the bladder gradually fills with urine, the detrusor muscle fibers of the wall of the bladder at first contract and then relax to accommodate the greater volume. The detrusor muscle, a smooth muscle, has the inherent ability to maintain tone and resist stretch.

A certain point of filling is reached when distension and the desire to void activate the sensory stretch receptors of the parasympathetic endings in the detrusor muscle. The stimuli for urination are transmitted to the reflex center in the spinal cord. If the time and place are not proper, completion of the reflex act can be voluntarily interrupted. Voiding is postponed by inhibitory impulses from the cortical center and voluntary contraction of the external urethral sphincter. When urination is desired, the higher-

ANN H. SCHUTT *was assistant to the staff,* GORDON M. MARTIN *is with the Section of Physical Medicine and Rehabilitation, and* THOMAS L. POOL *is with the Section of Urology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.*

level inhibition is suppressed, and reflex detrusor contraction occurs smoothly and powerfully. Vesical pressure is increased, and the urine is squeezed into the proximal part of the urethra, setting up a sensory stimulus which activates the motor impulses to the internal urethral sphincter. The internal urethral sphincter, normally closed, now opens and urine enters the posterior part of the urethra. This action serves as a second sensory stimulus which relaxes the external sphincter, and urine is expelled from the bladder. Contraction of the abdominal and diaphragmatic muscles and relaxation of the perineal muscles also play a role in the voluntary act of voiding.[2–4]

TYPES OF VESICAL DYSFUNCTION

Atonic neurogenic bladder. In the stage of spinal shock, immediately after injury to the spinal cord or in the presence of lesions of the lower motor neurons involving the posterior sacral roots of the reflex arc, an atonic neurogenic bladder results.[4,5] The sensation of fullness of the bladder is absent. The internal sphincter remains contracted and somatic reflexes are lost. The detrusor muscle of the wall of the bladder loses its tone. The bladder becomes overfilled and increasingly stretched and distended. There is little or no increase in intravesical pressure on filling until the bladder is overdistended. Overflow incontinence results as the mechanical pressure overcomes the forces of the external urethral resistance.[3,5] Potential or actual vesicoureteral reflux may occur because of decompensation of the ureteral musculature by repeated exposures to high intravesical pressures.[6] Within a few days to many months after the injury to the spinal cord, the patient gradually recovers from the spinal shock.[7] Quadriplegic patients often recover much sooner than those who have paraplegia.[5] Depending on the level of the spinal lesion, the bladder may come to be either automatic or autonomous.

Autonomous bladder. Lesions affecting both afferent and efferent limbs of the reflex arc leave the bladder as a completely autonomous organ.[4,5] This occurs after injury to the sacral spinal segments, with destruction of the reflex arc for micturition. The autonomous bladder is capable of exerting some expulsive force. Any contraction of the wall of the bladder is the result of impulses transmitted through the vesical ganglionic plexus which lies within the wall of this structure. Such contractions are of insufficient strength or duration to empty the bladder efficiently. Evacuation of the bladder often is accomplished by abdominal straining and manual compression, such as the Credé method.[3,5]

Automatic or spastic reflex bladder. The state known as automatic bladder or spastic reflex bladder occurs when the injury is located above the undamaged sacral center for micturition. The normal capacity of the bladder may be considerably increased in the early stages. The capacity eventually becomes reduced. The patient has no control over the act of micturition. Intravesical tension is increased and frequency may be noted, but often the quantity of residual urine is high. During the intervals between voiding, there is usually no leakage of urine.[2] The muscle wall can become hypertrophied and trabeculated as a result of the abnormal activity. Spasticity of the external sphincter usually is present, coincident with spasms of peripheral muscles. Eventually the bladder becomes small and contracted, with fibrosed walls.[1]

Regardless of the level of the injury, complete transection of the spinal cord is followed by inability to recognize the normal impulse toward urination. A vague sensation of fullness may remain in the form of burning or ill-defined painful sensations in the suprapubic area or the urethra.[2] When lesions are situated above the fifth thoracic segment, and especially when they are inflicted high in the cervical area, sensations of fullness completely disappear. These are replaced by manifestations of autonomic control characterized by flushing of the face, profuse sweating, chilling, headaches, "goose pimples," and paroxysmal hypertension.[2,4,8]

If transection of the spinal cord is partial, a wide variety of vesical dysfunction can obtain. Voluntary neurogenic bladder can develop, although inefficient vesical function may persist. Usually there is some degree of sensation and voluntary motor power when partial transection of the spinal cord has occurred. Vesical function may be normal or almost normal, with some dribbling of urine or stress incontinence.[2] The bladder of such patients may become small and spastic, associated with spasms of the lower extremities.[9]

Uninhibited neurogenic bladder. Uninhibited neurogenic dysfunction of the bladder rarely ensues after traumatic or acquired lesions of the spinal cord. This type of vesical dysfunction commonly results from damage to the cerebral cortex, caused by a cerebrovascular lesion, tumor, or partial destruction of spinal-cord pathways, by multiple sclerosis, or by disseminated sclerosis. Sensation is intact. As a rule, great urgency is coupled with inability to inhibit contraction of the bladder. This results in incontinence. The lack of cerebral inhibition, caused by cerebral

disease or damage to the spinal cord, is the fundamental factor underlying this type of vesical dysfunction.[5]

MANAGEMENT OF THE BLADDER

Initial measures for management of the bladder among patients with myelopathies can help to prevent complications which may involve the urinary tract. There is little disagreement about initial therapy. As soon as possible after injury, a No. 16 or 18 latex rubber indwelling catheter should be placed in the bladder with closed continuous aseptic urinary drainage.[8] Tidal drainage can be used, but, if it is, very close supervision is necessary. When tidal drainage is instituted, mechanical failure and damage to the bladder by overdistension can occur. A high intake of fluid, forced diuresis, acidification of the urine, use of a full diet, correction of anemia, two-hour turning of the patient, and physical therapy should be employed early.[5,8,10] Genital cleanliness should be maintained. Periurethral encrustations secondary to mucosal reactions to a catheter should be removed promptly, for these may contribute to the formation of urethral abscesses.[11] The catheter should be irrigated with a dilute antiseptic preparation, such as a 2% solution of boric acid, a 0.25 to 1% solution of acetic acid, a 1:5,000 solution of benzalkonium chloride (Zephiran®), or an isotonic solution of sodium chloride, at least two times daily, to eliminate debris formed in the catheterized bladder. The catheter should be changed every seven to ten days, or sooner, if the drainage seems sluggish. It has been suggested that the catheter be taped to the patient's abdomen to prevent erosion of the urethra by pressure of the catheter on the pubic bone.[12,13]

How long catheter drainage should be continued requires careful clinical consideration. When there is evidence of neurologic recovery, the catheter may be clamped. The bladder may be allowed to fill, and drainage may be accomplished at regular intervals, either hourly or every two hours. Continuous catheter drainage is maintained at night. When evidence of recovery of spinal-reflex activity appears, the catheter may be removed for a trial period of two to four hours. The volume of urine voided spontaneously should be noted. The catheter is then replaced and the residual volume is determined. By this method, vesical compensation can be estimated. Band[8] wrote that even though evidence of recovery of compensated function may be at hand, it is unsafe to discard the catheter suddenly. He preferred a progressive increase in the periods during which the patient is free of the catheter. Use of the catheter is discontinued when the volume of residual urine after a twenty-four-hour trial period is less than 100 cc.

During this time, the patient should be instructed to void on a set schedule and to regulate the intake of fluids. Usually, a regular routine for the intake of fluid during the day and the withholding of liquids from 7 p.m. to 7 a.m., employed with a system of periodic voiding, help to keep these patients dry. The patient may discover certain trigger areas on the abdomen or thigh which, if touched or stroked, will precipitate reflex micturition. The patient also can be instructed in the use of abdominal straining and the Credé method to achieve effective emptying. These procedures should be part of the training in self-care for the patient.

In some cases it will be impossible for the patient to develop satisfactory control of urination. In such instances, rubber-collecting appliances and pads can be used. Several surgical procedures, among them resection of the vesical neck, have been developed which can be performed to overcome specific problems.[14] Suprapubic cystotomy is rarely necessary or advisable.

Resection of the vesical neck. All patients with neurogenic bladder must be considered candidates for resection of the vesical neck, but decisions as to which patient will benefit by such a procedure calls for the exercise of fine judgment. Patients whose bladders are small and contracted and those who are incontinent are best treated by means of catheter drainage or external drainage, depending on the patient at hand.

When the decision to resect the vesical neck has been reached and the patient has accepted the decision, he must be told that multiple operations may have to be done. At the first operation, small sections of tissue should be removed from around the entire vesical neck. In years gone by, only tissue from the upper half or the lower half of the vesical neck was removed at the first step. This measure was so unsuccessful that we now advise resection of the entire circumference of the vesical neck.

In the postoperative period, the catheter is left in the urethra for four full days; then it is removed and the patient is asked to try voiding in a urinal. If this cannot be done, the catheter is replaced at the end of eight hours. If the patient voids in small amounts, the volume of residual urine should be ascertained accurately, and the catheter should be left in place if the volume of residual urine is more than 200 cc.

If the patient cannot void or empty the blad-

der well, another resection of the vesical neck may be considered. In the female, the absence of anatomic landmarks in the midportion of the urethra presents the danger of resection carried out too widely. Only occasionally can the fibers of a sphincter be discerned. In the male, the verumontanum is the external landmark, and resection of tissue should not be carried beyond that point. It is wise to remove too small, rather than too large, an amount of tissue at any one procedure. Incontinence can be a formidable complication if resection is carried out too widely.

Complications. Infection of the urinary tract is perhaps the most common complication and the first problem to be manifested among patients with myelopathies. The presence of a catheter within the bladder and urethra and the necessity for irrigation and changing of the instrument may introduce organisms into the bladder despite the most careful precautions to maintain asepsis. Efforts to eradicate these organisms from the urinary tract generally are unsuccessful in the presence of the catheter or other foreign bodies.[5]

Infection is the forerunner of nearly all other complications in the lower part of the urinary tract. Introduction of the urethral catheter also produces some degree of urethritis and cystitis. When infection has developed in the urethra or bladder, it also commonly extends into the prostate gland and epididymis.[15]

Periurethral abscesses, urethral diverticula, and urethral fistula develop fairly frequently; often they are associated with the use of large catheters.[11]

The management of urinary infections depends on the individual situation. Decision as to the appropriate antibiotic agent to use will have to be based upon results of culture of specimens of urine, studies of stained preparations of specimens of urine, and studies of the sensitivity of the organisms present to the agent in question.[15] The intermittent or continuous administration of such nonantibiotic antibacterial agents as sulfonamides and methenamine mandelate (Mandelamine®) and of such acidifying agents as methionine may be indicated for certain chronic infections. Suppressive therapy often is used hopefully to reduce progressive renal damage when a chronic urinary infection is present. Nonantibiotic antibacterial agents may be given prophylactically for one week of every month or every other week.[16]

Stasis in the lower part of the urinary tract, which harbors residual urine, also invites infection. Infection may reach the kidney by hematogenous or lymphatic routes or by direct retrograde inoculation as a consequence of ureteral reflux. Episodes of acute pyelonephritis increase in frequency and severity when residual urine is present. Each attack of pyelonephritis may compromise renal function.

Formation of stones in the bladder or kidney is the next most frequent complication among patients with myelopathies. This condition is particularly troublesome during the first 2 years of the illness. Hypercalcinuria and hyperphosphaturia, coupled with changes in protein metabolism, are associated with long periods of recumbency and lead to subsequent osteoporosis. The increased urinary excretion of calcium and phosphorus increases the tendency for these minerals to precipitate out in the urinary tract. Infection produces an additional stimulus to the formation of calculi and is most common in the presence of urea-splitting organisms, which bring about alkaline urine.[2,5,15]

Hydronephrotic dilatation of the upper part of the urinary tract is another frequent complication. Hydronephrosis, which complicates the neurogenic bladder, is caused by vesicoureteral reflux, diminished ureteral flow from obstructing calculi, or stricture of the ureterovesical junction. Elevated vesical pressure, pooling of urine in the bladder, and infection also can lead to hydronephrosis.[2,5] Reflux of urine from the bladder is common among patients who have lesions of the lower motor neurons. Periodic excretory urograms are necessary for determination of this complication.

The bladder may become contracted and affected by prolonged suprapubic drainage, infection, calculi, and fibrosis.[17] Spasticity may develop as a result of hypertonicity of the musculature of the bladder and excessive reflex activity elsewhere, such as in the mass reflex activity of the lower extremities. The consequence of this abnormal activity is that the muscular wall becomes hypertrophied and trabeculated. Similar changes take place in the sphincters. Eventually the bladder becomes small and contracted, with fibrosed walls. This situation is virtually irreversible.[1]

It is evident that good initial urologic treatment is essential in the prevention of many of these complications. Dilatation of the upper part of the urinary tract was found in 70 of the 184 paraplegic patients whose condition was followed by Damanski from 1947 to 1960.[18] He found that 22 of 28 patients who had had inadequate management of the bladder in the early stages of the paralysis had died. In a second group of patients who had received adequate

initial treatment, 5 of 40 had died of uremia. Damanski noted that the number of patients who died of uremia was greatest in the sixth posttraumatic year and that another increase, but a lower one, appeared in the tenth year. He wrote that hydronephrosis must be eliminated or death from uremia will follow. Only prolonged periodic follow-up studies, urologic examinations, and frequently made excretory urograms will reveal these late complications.

NEUROPHYSIOLOGIC ASPECTS OF THE LOWER BOWEL

The innervation of the large bowel, sigmoid, and rectum is similar to the innervation of the bladder. Only one important difference exists: There is no voluntary control over the contractions of the bowel and rectum. Usually the feces are stored in the pelvic colon until defecation takes place. At the junction of the pelvic colon and the rectum there seems to be an obstacle to the progress of feces. When this valvelike mechanism is overcome by mass peristalsis, the feces move into and distend the rectum. This distension stimulates a perineal sensation which is interpreted as the desire to defecate. Defecation is a reflex action. Distension of the rectum provokes reflexly a strong wave of mass peristalsis and contractions of the rectum. When rectal contraction occurs, the internal and external anal sphincters relax. The large intestine from the transverse colon downward is then emptied. Emptying of the colon is aided by the simultaneous contraction of the muscles of the diaphragm and abdomen, the levator ani, and the other perineal muscles. Only the external anal sphincter is under voluntary control and can inhibit this reflex.[19]

INTESTINAL FUNCTION IN PATIENTS WITH MYELOPATHIES

The mechanism of defecation is depressed by spinal shock after injury to the spinal cord. During this period the sphincter ani relax only slightly.[19] The tone of the sphincter muscles generally begins to recover within a few days. After several weeks to several months, the rectum may begin to empty itself automatically.[20,21] When sacral innervation is destroyed the rectum contracts, and reciprocal relaxation of the anal sphincter takes place. The contractions of the rectum appear to be related solely to the myenteric nerve plexus. As is the process in the normal person, the mechanism of defecation depends upon the reaction of the rectum to distension and adequate expulsion of fecal material from the colon. The effect of the reaction to distension of the rectum upon the sphincter ani is twofold: an immediate relaxation of the sphincter and a delayed resumption of tonic contraction of the sphincter. The passive tension in the rectum appears to be sufficient to bring about relaxation of the sphincter ani, but active tension is a more powerful stimulus.[19] Cutaneous stimulation in the area of the sacral segments, particularly around the anus, and activity of the bladder both facilitate the occurrence of reflex defecation.[19,21]

Management. For the treatment of patients in spinal shock, enemas and laxative agents may be necessary. Soon after the period of spinal shock, the change from the use of enemas to the status of automatic defecation may require only a few days of training. Pollock and associates,[21] using barium meals, found that there was no impediment to the flow of barium as far as the large bowel in these patients. But, when charcoal meals were employed, they found that passage of the stool along the large bowel and the rectum was greatly delayed. They noted that after the first appearance of charcoal a number of days could elapse before the charcoal completely disappeared from the stool. They also concluded that intestinal action depends considerably on the consistency of the stool. After long retention, the stool becomes formed, dehydrated, and hard so that obstipation develops. Incontinence results, with loose stools. An ideal fecal consistency may be obtained by the use of fruit juices, a high intake of water, and, when necessary, the use of stool softeners, such as wetting agents or hydrophilic colloids.[5,21]

In the developing of reflex defecation, a regular time period should be set aside for this program, either daily, every other day, or every third day. When possible, a relaxed sitting position is best for emptying the bowel. A stimulus designed to stretch the anus may be necessary. This may be accomplished by the use of glycerin suppositories, bisacodyl (Dulcolax®) or Vacuetts suppositories, or by the patient's wearing a rubber glove and manually stretching the anal sphincter. As the tone of the rectal muscles returns, the patient may note greater ease in producing a bowel movement, and less time will be needed to stimulate the rectal contractions. The patient should be encouraged to use abdominal muscles and to strain moderately. If these muscles are paralyzed, manual pressure can be exerted, in consonance with activation of the muscles of the diaphragm, to increase intra-abdominal pressure. As these manipulations become more efficient, eventually the patient may be able to induce bowel movements merely by

touching the area of the anus; then use of the external stimulus may be discontinued gradually as the patient regains automatic defecation with the help of mild-to-moderate straining.[5,21]

Complications. If enemas are relied upon for evacuation of the bowel, a larger staff of attendants will be necessary. Medical care and therapy are impeded. The morale of the patient may be low. An enormous quantity of supplies and linen will be required. Patients who have to depend upon enemas for evacuation of the bowel develop fear of soiling when traveling or on leave from the hospital. They lack confidence in control of the bowel. The use of enemas is inconvenient and wastes time.

The adverse effects of reliance upon enemas include the symptoms and signs of disturbance of other functions. Patients who have lesions in the cervical and upper thoracic parts of the spinal cord may experience increased autonomic reflexes. These reflexes may result in hypertension, headache, profuse sweating of the face, chills, dizziness, and, possibly, loss of consciousness. Nausea, and, at times, vomiting, abdominal discomfort, anorexia, increase in spasms, and burning pain are common among patients with lesions at any level of the spinal cord who require enemas for evacuation of the bowel. The patient may be more or less harnessed by the effects of these daily enemas.[21]

Fecal impactions are common among these patients, particularly those who have been injured more recently, but impactions can occur at any time. An inadequate intake of fluid or food, irregular bowel habits, inactivity, and inattention to intestinal function may lead to fecal impactions. These, in turn, may lead to perforation and obstruction of the bowel and, occasionally, to the frequent formation of watery stools around the site of impaction. Hemorrhoids, which cause rectal bleeding and resultant anemia to some degree, may be a complication of routinely hard or impacted stools. Perforation of the rectum is an ever-present danger when enemas are used or instrumentation is employed. The formation of abscesses and fistulas and the development of rectal stricture and septicemia may occur secondary to perforation.[21]

The continued use of laxative agents can produce an inactive bowel in an already sluggish mechanism and also can generate a sense of insecurity due to the fact that incontinence may occur at any time. There is always the possibility that infection of the bladder may be caused by bacterial contamination with the contents of the bowel.[21]

SUMMARY

The problems and complications involving the bowel and bladder of patients who have myelopathies are many. Adequate management of the bladder and bowel, as part of the therapy and rehabilitation of these patients, may mean the difference between a prolonged and relatively comfortable life and a shortened survival with many illnesses.

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Perceptual Problems of the Hemiplegic Patient

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THE HEMIPLEGIC PATIENT has a prominent and readily noted motor defect, but there may be subtle perceptual losses that are easily overlooked. The loss of ability to make sense out of sensations may slow progress in rehabilitation more than the paralysis, causing frustration to the patient, the physician, the therapist, and the family. Many patients with perceptual problems are unjustly labeled as being poorly motivated.

Perception is the ability to sum up and integrate all the myriads of incoming sensory messages. This phenomenon is accomplished by the smooth working of the whole nervous system, but most neurologists agree that a large share of the responsibility falls to the parietal lobes.[1–3] In man, the parietal lobes are prominent, extending from the fissure of Rolando back to the parieto-occipital fissure and from the sylvian fissure upward to the midline and down into the longitudinal sulcus to the body of the corpus callosum. The major portion of the blood supply to the parietal lobe is via the middle cerebral arterial system, a complex commonly involved in cerebrovascular accidents. In addition, the anterior cerebral artery supplies the anterior and medial portion of the parietal lobe, and the posterior cerebral artery sends a branch above the calcarine fissure to the posterior portion of the parietal lobe. It is possible, therefore, to have damage to the parietal region when occlusions occur in any of the three major arterial systems to the brain.[4]

RECOGNITION OF PERCEPTUAL PROBLEMS

Since, for any activity to be meaningful, it must be correctly related and correlated to the current situation, it is important for the physician to determine if the patient is actually making an adequate analysis of the external world. Such an assessment is difficult for several reasons. Often, the patient with a defect in the parietal lobe is a poor observer, has marked fluctuations in mental ability, has a short attention span, and is highly suggestible. Testing periods must be brief, and frequently the tests must be repeated. Great care in testing is necessary to avoid inadvertently pointing the way to certain answers. Visual and communicative problems frequently arise and may necessitate alterations in the testing methods. Further, the examiner faces an overwhelming number of sensory perceptions that could be tested; in fact, ingenuity has been taxed severely just to name all of the various perceptual phenomena. Consequently, it is difficult to devise tests that are specific and to record the results in an understandable and standard way.

Elaborate and complex tests are being used in research projects, but this paper concerns the more common, simple tests that can be used during a neurologic examination.

Perception testing. Hemiplegics may show some unilateral loss of tactile sensibility when tested with a wisp of cotton. This hypesthesia may be unevenly distributed over half of the body, and the hand is often the most severely affected part.[5] The loss will not follow a dermatome or peripheral nerve pattern, of course, and its borderlines are indistinct. The medial border often falls slightly short of the midline of the body. Areas of insensibility may be established by suggestion, or even repetition of tests may be sufficient to cause iatrogenic hypesthesia. The patient will seldom show concern about his sensory loss, and he may have illusions or hallucinations of touch. The touch sensation may be transferred and perceived as coming from the opposite side. Localization and discrimination of fine differences in touch, pain, and temperature are especially difficult for patients with parietal lesions.

Two-point discrimination or the compass-test is often useful in testing the integrity of the parietal lobe. The ability to recognize the duality

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of two points, simultaneously placed in close proximity to each other, varies considerably from one region of the body to another. The lips may discern the stimuli as arising from two points when they are only 2- to 3-mm. apart, while the shins may require separation of at least 4 cm.[6] Ability will vary in different individuals and will also improve with the training acquired in previous testing sessions. There is a marked tendency for patients with parietal disease to give irregular, incongruous, and unpredictable replies. In detecting the perceptual loss, it is important to compare one side of the body with the other—that is, to compare the patient's responses to stimuli applied to the two sides.

Faulty recognition of passive movements of an extremity is common in the presence of parietal lesions. This may be manifested by unawareness of the direction of movement and the position of the limb in space. It may lead to wandering, so-called pseudoathetoid movements of the extremity. Also, it may explain some of the peculiar, uncomfortable positions that a patient will allow his arm to take while he is lying in bed. There may be an alteration in the subjective sensation of the weight of the extremity. Position sense of the skin (dermatokinesthesia) may be checked by picking up a fold of the patient's skin and displacing it either up or down or right or left and questioning the patient as to the direction of movement. It seems evident that if the summation of body movements is inadequately perceived, it would not be possible to construct an accurate "schema" or postural model of one's self. Meaningful action must be based on some conception of where the body and limbs are.

To be able to identify an object from taetile stimuli is an important function. Vision is generally used together with touch to provide identity to objects in our environment. The mind is generally thought to picture objects and we speak of the "mind's eye" and of "visual memories," but other modalities of sensation are closely interwoven. As an example, if one should spot a wet dog running across the highway, the visual stimulus would be enough to "feel" its wet fur, to "hear" its panting, and to "smell" its odor. On the other hand, if one were blindfolded and led up to a wet dog, it would be necessary only to take one sniff or lay a hand briefly on the dog's back to conjure up the image of a wet dog. Patients with pereeptual problems may have trouble putting the mental image together and thus miss identification of common objects.

Stereognosis, or recognition of objects by tactile sensation alone, is largely a complex relationship of two-point discrimination and postural sense referred to memories of past experiences.[2] If touch discrimination is poor, the molecular qualities of objects—that is, smoothness and hardness or density—may not be perceived; this is called "ahylognosia." If postural perception is poor, it may be difficult to recognize size and shape (amorphognosis). When the patient cannot recognize an object and there is neither amorphognosis nor ahylognosia, the defect is called "tactile asymboly."

When testing for astereognosis it is best to record not only that the correct identity was or was not made but also whether there was prolonged searching. Subtle losses may be detected by having the patient wear gloves during testing. Placing objects in both hands simultaneously may reveal that one hand is much slower than the other.

Objects may be misjudged as larger than they are (macrostereognosia) or smaller (microstereognosia). The contour may be changed and distorted in perception (metamorphotaxis). Errors may be made in estimating the weight of objects (abarognosis).

Perceptual difficulty can be observed by stimulating a patient in two widely separated areas and noting if both stimuli are discerned. Commonly the patient is given brief light touches simultaneously on the faee and hand and asked to report where he felt the stimuli. The patient having a parietal lesion may indicate that he felt only the touch on the face. There seems to be some order of preference, the face stimulus being most often noted and the hand stimulus ignored—that is, the more rostral of two sensations will usually dominate over the more caudal.[5] This general scheme of simultaneous testing is not limited to touch alone but can also be used for testing visual, stereognostic, barognostic, and morphognostic problems. Various modalities ean be mixed together in testing; this may show, for example, that touch is ignored when the patient is simultaneously stimulated by a light or sound. If the stimuli are on opposite sides of the body, the stimulus on the hemiplegic side is usually ignored. Sensation may be transposed from the affected side and perceived as on the normal side (allochiria). The reverse phenomenon has been recorded, where objects being held in the normal hand seem to be contained in the abnormal hand.[1]

Usually, with problems related to double simultaneous stimuli, there is difficulty in loeating the stimulus accurately on the affected side. This displacement of the locale of a stimulus is usually to a rostral part of the extremity

but may be even to a point out in space (exosomesthesia) or perhaps to a place where the limb had been previously.

PERCEPTUAL PROBLEMS

Perception of self and illness. One of the most curious phenomena associated with parietal lesions is the ability of the patient to ignore and neglect his affected side. This occurs in various gradations of severity. A patient may show only passive disregard for the affected side. This may be shown by asking the patient to raise his hands. He may raise only the hand on the intact side, but when reminded that the other hand was not raised, he will lift it immediately.

Despite the fact that half of the body is paralyzed, the patient may show an amazing lack of concern; this phenomena is called "anosognosia." Patients may actively deny the existence of paralysis despite a demonstration of inability to move the affected part. The patient may state that he broke his arm when a child and that it has always been weak or that he often does not have strength in the morning. This type of reasoning seems adequate to the patient to explain calamitous symptoms.

Occasionally, the patient will attribute the affected arm with a personality of its own, giving it a name and talking to it. There may be erotic feelings between the patient and his arm. The patient may feel that the limb belongs to another person (somatoparaphrenia).

At times the patient believes there is nothing remaining on the affected side (asomatognosia).

If a patient is tactfully confronted with the facts, he may switch from one degree of acceptance to another. The truth may be accepted with an unexpected light-hearted attitude.[1]

Perception of space. Visual defects and visual perceptual loss may occur when there is thrombosis of the basilar, posterior cerebral, middle cerebral, or strial arteries or, occasionally, the anterior choroidal arteries.[3,4] Central or cortical blindness may result with the peculiar feature that the patient will not complain of being blind. He may make excuses for his clumsiness and not use the logical precautions that blind people take when walking. Blindness may not be complete, and partial recovery may occur.[1]

With a unilateral lesion of the posterior half of the brain, hemianopsia occurs. Central vision or the fixation point is often spared. If homonymous hemianopsia occurs because of an anterior lesion behind the chiasma, the patient will compensate by swinging his eye back and forth and tilting his head so that the blind field is explored with the better visual half. In contrast, the patient with a parieto-occipital lesion has an imperfect awareness of his disability; he perceives objects as being cut in half, and he continually bumps into objects on his blind side. It is important that the patient's bed be so placed that his intact visual field faces the room, rather than the wall, and that doctors, nurses, and visitors approach and remain on the side which can be perceived by the patient.

Hemiplegic patients have difficulty in judging vertical and horizontal relationships and whether an object is straight ahead.[7] When required to judge when a luminous rod is vertical in a darkened room, hemiplegics make a significantly greater number of errors than do normal persons. Patients having left hemiplegia make greater errors than do those having right hemiplegia. Birch and co-workers[8] noted that errors both for vertical and for horizontal vision were counterclockwise, meaning that the entire visual field was rotated. Judging the vertical in a dark room is not just a matter of vision but also involves analysis of proprioceptive stimuli from the neck and body and vestibular centers. If the lights of the room are turned up or the light is rotated about in a stationary rectangular frame, the hemiplegic may still make more errors than the normal person, but, in general, he does much better than in the dark. This perceptual problem may partially explain the tendency of a hemiplegic patient to lean to the affected side. Undoubtedly, it is not always due entirely to weakness; rather, it may relate partially to his attempt to align with his perceived vertical.[9] Walking in well-lighted areas may help to correct this problem. Vertical imperception would add greatly to the danger of falling at night on the way to the bathroom. In perceiving whether an object is straight ahead, Birch and co-workers[7] found that patients having left hemiplegia had greater trouble than did those having right hemiplegia and that judgment was improved in the light.

Patients may have trouble judging the size of objects which may appear to be distorted, seeming larger or smaller in one or two dimensions. Objects may even be inverted. Stereoscopic vision may be lost. Stationary objects may have apparent movement or moving objects may appear to move faster or slower than they do. There may be perseveration of images so that objects are seen more than one time. All of these alterations in perception would place giant hurdles in the path to satisfactory ambulation.

The art of spatial revisualization may suffer so that the patient cannot draw a map of his country or neighborhood or sketch the location of the furniture in his own room. He may re-

peatedly stumble over familiar objects in his environment or get lost on the street where he has lived all his life.[1]

Misconceptions of body image and space are often portrayed in pictures that patients draw. When drawing a man, the affected side may be incomplete or even missing. The face of a clock may be lopsided with half the numbers missing. Or, the patient may go into a "sorcerer's-apprentice syndrome" of drawing the same small detail over and over again, at a faster and faster rate.[10] Handwriting may be cramped to one side and written askew. In attempts to reproduce matchstick figures, not only does the patient make errors but also he may lay his pattern directly over the model or borrow matchsticks from the model pattern to complete his own pattern. Working with form boards or building blocks, patients show problems in three-dimensional construction. Various dyspraxias are not the subject of this paper, but they certainly would blend in with the patient's distortion of space perception.

COMMENT

It has been noted that patients having left hemiplegia have more perceptual problems than do those having right hemiplegia. It has been suggested that this results because patients having right hemiplegia often have language problems and disorders of symbolic thought so that their problems are simply not communicated to the examiner. Language may be impaired by lesions of the dominant side, but there remains a controversy as to whether body image impairment occurs solely or even chiefly from lesions of the nondominant side.[11] The left side of the body in most people is normally subordinate to the right and, if it becomes affected, it may be more readily ignored.

Carroll[12] and also Karlin and Hirschenfang,[13] in measuring visuospatial impairment, found more difficulties in judgment, calculation, and time awareness in the left hemiplegic. These defects did not respond to therapy and so these patients were less successful in vocational rehabilitative efforts. Knapp,[14] in a small series of patients, showed a poor record in returning left hemiplegics to work. The left hemiplegic may look deceptively competent, being able to talk clearly and to have independent ambulation, but actually he may be less able to assume responsibility because of fluctuating judgment, inability to copy, short attention span, and poor retention of facts.[12,15] Mistakes may not be realized even when they are plainly pointed out to the left hemiplegic.[11]

SUMMARY

When a physician treats a hemiplegic patient, he wants him to achieve maximal rehabilitation. Sometimes desired functional independence may not be achieved because of a severe motor deficit; however, a significant number of patients do not fully succeed in rehabilitation because of perceptual losses. Neurologic tests of sensation and coordination may reveal perceptual problems which can be the basis for delayed and limited success in rehabilitation. If a patient cannot correctly analyze stimuli from his skin, joints, or eyes so that he can accurately visualize himself or the space around him, he has a handicap as great or greater than his motor paralysis.

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Physiology of Balance

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For the patient who has suffered damage to the central nervous system, the problem of restoration of balance for standing or walking may be difficult. Often, prolonged training using every available technique to regain muscular control is necessary to achieve limited standing and ambulation. Successful rehabilitation of this type is based upon utilization of the residual physiological reflex responses of the central nervous system. Therefore, a knowledge of the physiology of the neuromuscular system is necessary as the basis for a therapeutic program.

As a preliminary to considering the physiology of balance, it should be emphasized that under normal circumstances standing with minimal expenditure of energy is achieved by positioning the body in such a way that muscles need not support the weight of the body. During relaxed standing, the knee, hip, and lumbar spine are held extended against the restriction of ligaments and fascia on the flexor side of each joint by the force of gravity without muscular action (Fig. 1). If contractures develop so that normal full extension cannot be achieved, great muscular effort must be expended to maintain the erect position. Even persons with normal strength cannot stand for a prolonged period of time in a position which requires that muscles support the weight of the body. Too much energy is required. Fatigue occurs quickly and the person is forced to sit or lie down.

Most of the balance of the trunk, flexible as the spine may be, is accomplished by positioning the vertebrae in such a way that the spinal ligaments support the upright position. During relaxed standing, the anterior spinal ligaments restrict extension of the lumbar spine, and the posterior ligaments restrict flexion of the thoracic spine as the person slumps into a kyphosis. In this relaxed posture a person "hangs on his ligaments" with minimal muscular effort.

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The physiology of normal balance involves the integration of multiple reflexes at multiple levels of the neuromuscular system. The volitional control of the muscular system has as its basis the utilization of many spinal and supraspinal reflexes. In the normal individual it is difficult to isolate the reflexes of the various levels in the central nervous system. In patients who have suffered damage to the central nervous system, the modulating influences from the upper neural centers may be cut off, leaving the reflex responses uncontrolled. To simplify the understanding of the reflexes involved in neuromuscular control, they are presented here in relation to the levels in which the reflex centers lie.

Spinal Reflexes

Let us first consider the reflexes of the spinal cord. The reflex arc contains sensory receptors at which an impulse originates, a sensory nerve fiber leading to the spinal cord, synapses between two or more neurons within the spinal cord, and a motor neuron with its axon conducting nerve impulses to the muscle fibers of the motor unit. The sensory endings may arise either in the skin, responding to the stimuli of pain, temperature, and touch, or from proprioceptors in the deeper tissues sensitive to pain, pressure, and tension (Fig. 2). The reflex may be monosynaptic, such as the stretch reflex, with the axon of the sensory neuron synapsing directly with the motoneuron in the anterior horn of the spinal cord. Reflexes may also be multisynaptic, with the axon of the primary sensory neuron synapsing on dendrites of an internuncial neuron. There may be one or more internuncial neurons in the reflex chain before a synapse is finally made with the motoneuron. The more complex the chain of internuncial neurons, the greater is the possibility for modification of that reflex.

Impulses entering the cord through the sensory pathways may also go to higher centers through internuncial pathways. These may extend to reflex centers in the medulla, the mid-



Fig. 1. Normal relaxed standing posture behind a vertical grid showing the maintenance of position by the force of gravity holding the lumbar spine, hip, and knee extended against restricting ligaments

brain, the cerebellum, and the basal ganglia or may be perceived when connections activate the cerebral cortex. Neuroactivity initiated at any center may result in discharges back to the spinal cord which may augment or inhibit the spinal reflexes. It must be emphasized that instead of the oversimplified concept that a single impulse or very few impulses from a sensory neuron impinging on a motor neuron cause it to discharge, many impulses must impinge upon the anterior horn cell and its dendrites nearly simultaneously in order to raise its level of excitation and cause it to discharge an impulse down the axon to the motor unit (Fig. 3).

Neurological studies indicate that there are two types of effects of nerve impulses influencing the activity of a motor neuron. There are excitatory impulses from many sources which synapse on the dendrites of the motoneuron. These impulses coming in simultaneously from many sources increase the excitability of the neuron which at a certain level discharges an impulse into the axon. At the same time there are impulses through other connections to the motoneuron which produce inhibition to motoneuronal activity. How this is accomplished is not understood. It has been postulated by Gesell and Dontas[1] that the spatial relationships of the synaptic connections on the motoneuron determine whether an impulse is excitatory or inhibitory. It appears that synapses on the neuron close to the axon hillock decrease the excitability of the motoneuron, while more distal synapses on the dendrites increase excitation. The balance of excitatory and inhibitory impulses arriving at an anterior horn cell will determine whether or not the level of activity increases enough to discharge an impulse through its axon to the motor unit, thereby causing a muscular contraction. The flow of impulses through the multiple connections of the millions of internuncial neurons

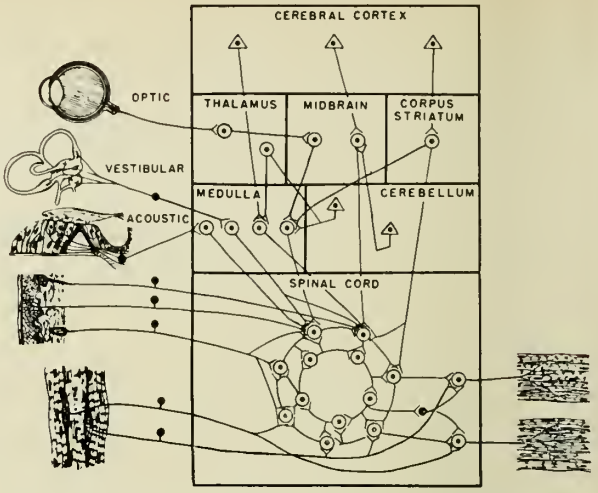


Fig. 2. Diagram of activation of the internuncial pool of the spinal cord by sensory inflow from cutaneous or proprioceptive sensory neurons, special sensory receptors, or impulses from higher centers. Motor responses depend upon continual activity of the internuncial pool.

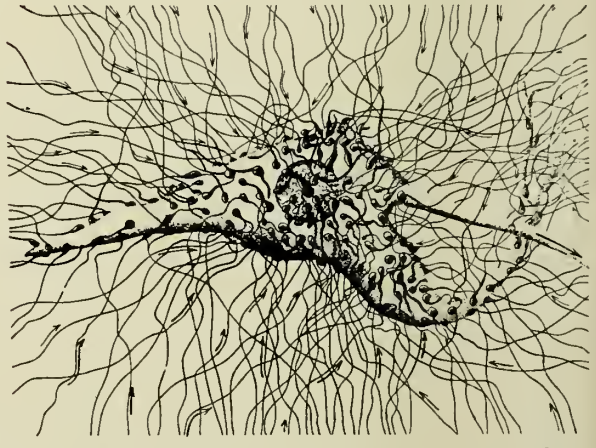


Fig. 3. Motor neuron showing the many synaptic endings connecting to it (from Katz, Scientific American 205:209 [Sept.], 1961).

in the central nervous system results in a high level of activity within the central nervous system and a certain level of excitation of all anterior horn cells at all times. Volitional excitation of motor function from the cerebral cortex merely increases this level of activity very slightly so that it exceeds the threshold necessary for an axonal discharge. Without the basic activity which is dependent upon the multiplicity of impulses from many sources, volitional activity would not be possible. This activity in the central nervous system is maintained by internuncial connections from sensory neurons stimulated through the superficial sensory endings in the skin; the proprioceptive endings in the fascia, muscle, bones, and joints; sensory endings in the

viscera; and special sensory endings such as the acoustie, vestibular, and optie receptors. All of these increase the level of excitation in the spinal cord so that very few additional impulses from the motor cortex will raise the level of excitability of the anterior horn cell above the threshold and eause a museular eontraction. The eoneept of an internuneial pool, postulated by Lorente de No,[2] as a eontinual flow of impulses through multiple internuncial circuits within the central nervous system helps to explain observations regarding control of the neuromuscular system. The importance of a basic high level of exeitation within the spinal cord to raise the exeitability of the motoneuron close to the diseharge threshold so that volitional impulses ean be effective is shown after injuries to the spinal cord, when the phenomenon of spinal shock occurs with loss of motor activity and flaccid paralysis. Eaeh of the various reflexes, therefore, shows its effect beeause of this background of activity in the internuncial pool of the spinal cord, and eaeh reflex becomes ineffective without such background aetivity.

The spinal reflexes that are involved in balance are listed in Table 1. The flexion reflex is a simple segmental reflex resulting in eontraction of the flexor muscles of a joint elose to the stimulus when the superficial sensory endings in the skin are stimulated. If the stimulus is increased, the loeal flexion reflex may spread to other muscles of the extremity, producing a more extensive flexor response.

In the ease of injury to inhibitory pathways, the resistanee to synaptic transmission in the spinal eord is decreased, and as the result of minor stimuli, impulses spread through many internuneial pathways to cause distant and local responses. Visceral reflexes as well as skeletal muscular reflexes may then be elieited. This is the meehanism of the mass spinal reflex which follows transeetion of the spinal eord. The flexion reflex interferes with stanee and balanee, because

TABLE 1

SPINAL REFLEXES INVOLVED IN BALANCE

Flexion reflex
Mass spinal flexion reflex
Stretch reflex
Alpha neuron reflex
Gamma neuron reflex
Crossed extension reflex
Tendon reflex (Golgi)
Extensor thrust
Long spinal reflexes

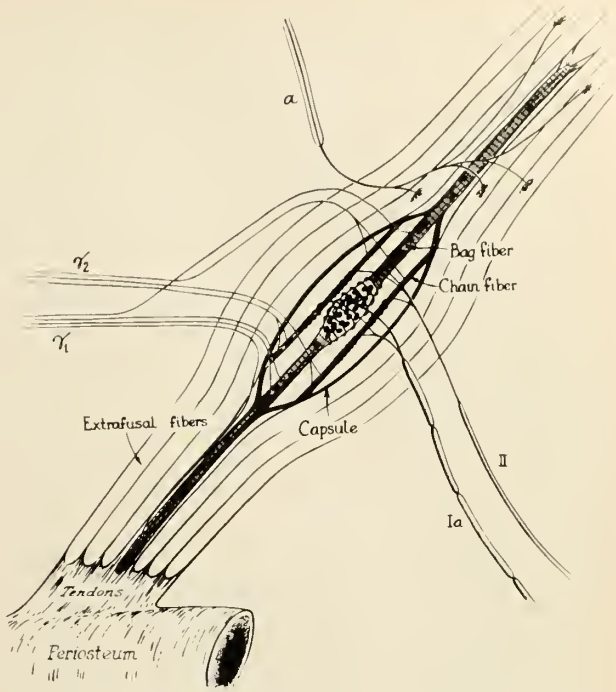


Fig. 4. Diagram of the structure of a muscle spindle. Stretch of the nuclear bag stimulates the primary afferent endings, Ia, causing the monosynaptic reflex contraction of the extrafusal muscle fibers through the alpha motoneuron. Contraction of the intrafusal bag fiber increases the stretch on the nuclear bag and increases the sensitivity of the stretch reflex. The motor innervation of the intrafusal muscle fibers is derived from the gamma motoneurons.

a stimulus initiating this response will eause flexion of the lower extremities with loss of the support of the body.

Another segmental spinal reflex is the streteh reflex, initiated by stretching the annulospiral or primary sensory endings on the intrafusal muscle fibers of a muscle spindle. Tension on the nuclear bag of the intrafusal musele fiber apparently is the effective stimulus initiating this reflex (Fig. 4). Stretch of the muscle spindle stimulates the diseharge of sensory impulses into the spinal cord, through a single synapse to the motoneuron, and results in a contraction of the stretehed musele and its synergists, together with inhibition of its antagonists.

The sensitivity of the streteh reflex is modified by a seeond reflex, the gamma neuron reflex. If the intrafusal muscle fiber of a musele spindle contracts, it stretehes the nuelear bag and inereases the sensitivity to stretch of the sensory endings located on it. Small, thinly myelinated axons from gamma motoneurons provide the motor innervation for the intrafusal musele fibers. The reflexes aetivating the gamma moto-

neurons, and thereby increasing the sensitivity of the stretch reflex, may be initiated or augmented from a number of sites. Stimulation of the skin overlying the belly of a muscle causes a gamma neuron reflex which results in contraction of the intrafusal muscle fibers and increases the sensitivity of the stretch reflex of that muscle. At the same time, these stimuli inhibit reflexes in the antagonistic muscles. In the gamma neuron reflex, impulses entering the dorsal root of the spinal cord are known to pass upward through the cord to centers in the high cervical region and the brainstem, where synapses occur and efferent fibers return down the cord to the gamma neurons in the anterior horn. Both the sites of initiation and the spread of the reflex response are much broader than in the case of the primary stretch reflex.

The crossed extension reflex is a more complex bilateral spinal segmental reflex. If, through cutaneous stimulation, a flexion reflex is elicited strongly on one side, there will be extension of the opposite extremity. Teleologically, this provides a mechanism for moving away from a noxious stimulus by providing support to the body and making it possible to withdraw the stimulated extremity by flexion. There is also a reciprocal reaction so that strong extension of one extremity invokes flexion in its contralateral member.

The tendon reflex of Golgi is a protective reflex preventing excessive contraction of a muscle. This reflex is stimulated by tension at the musculotendinous junctions where the specialized endings are located. In response to strong tension exerted on the muscle, there is inhibition of the contraction of the muscle, and the response to stretch reflexes or to volitional contraction is diminished.

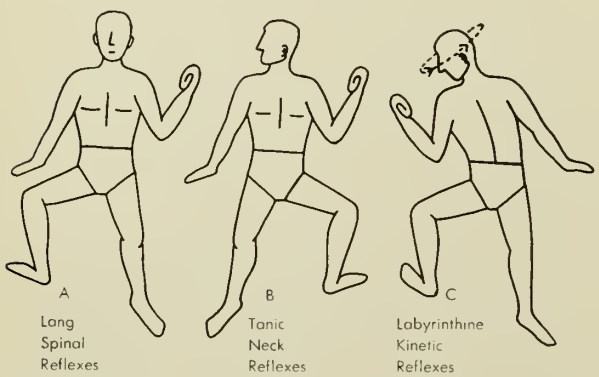


Fig. 5. Reflex postures. (A) Spinal posture from combined long spinal and crossed extension-flexion reflexes, (B) asymmetrical tonic neck reflex posture, and (C) labyrinthine kinetic reflex posture

TABLE 2

SUPRASPINAL REFLEXES INVOLVED IN BALANCE

Tonic neck reflexes
Labyrinthine reflexes
Static reflexes
Kinetic reflexes
Positive supporting reaction
Negative supporting reaction
Righting reflexes

A sixth spinal reflex directly related to postural tone is the extensor thrust reflex. If the flexors of the toes are stretched, increased tone develops in the extensor muscles of the ankle, the knee, and the hip.

The long spinal reflexes are intersegmental postural reflexes (Fig. 5). With regard to the position of any extremity, its ipsilateral member will assume the opposite position. When the long spinal and the crossed extension reflexes combine, the typical spinal figures are obtained. For example, if one arm is extended, the contralateral arm and the ipsilateral leg will be flexed, and the contralateral leg will be extended. These reflexes exert their influence both rosterally and caudally so that arm and leg positions influence the opposing member reciprocally. It is obvious that this reflex has an important influence on the pattern of reciprocal motion which occurs during walking. It has been shown that at least a part of the change in tone of an extremity due to the long spinal reflexes is mediated through the gamma fiber system.

SUPRASPINAL REFLEXES

The supraspinal reflexes are indicated in Table 2. The tonic neck reflexes influence the tone of muscles in the extremities and trunk as a result of the position of the neck. The stimuli are initiated at sensory endings in the joints of the first and second vertebral segments, and the sensory fibers enter the spinal cord through the first three cervical roots. The center for these reflexes is located in the reticular formation in the lower part of the medulla and the upper part of the cervical cord. These reflexes appear to influence the activity level of alpha and gamma motoneurons. Multiple responses occur from the tonic neck reflexes. When the neck is flexed in the sagittal plane, there is an increase of the flexor tone and a decrease of the extensor tone in the arm and hand. When the neck is extended in the sagittal plane, there is an increased extensor tone and a decreased flexor tone in the arm and hand. When the neck is rotated or tilted laterally, there is increased extensor tone in the chin, arm,

and leg, that is, the arm and leg toward which the chin is turned or the head is tilted and an increased flexor tone in the skull arm (Fig. 5). In normal individuals, the tonic neck reflexes may be clearly evident only when a person is performing against heavy resistance, under which condition it may be seen that the neck assumes a posture which will facilitate the activity being attempted. Thus, during flexion of one elbow against heavy resistance, the neck will be rotated and tilted toward the opposite side, and there will be increased extensor tone in the extremity on the opposite side.[3] It is probable that the tonic neck reflexes act mainly on the posture of the upper extremities and that usually the tone and position of the lower extremities are influenced secondarily through the long spinal reflexes.

The labyrinthine reflexes consist of two types—the static reflexes and the kinetic reflexes. The labyrinthine reflexes are postural reflexes which, together with the long spinal reflexes and the tonic neck reflexes, exert a profound influence on the posture and muscular tone of the extremities. The static reflexes have as their stimulus the pull of the otolith in either the utricle or the saccule. The reflex activity is maximal when a person is lying supine with the head elevated 45° and is minimal when a person is lying prone with the head dependent 45°. When a person is upright, the static labyrinthine reflex increases flexion in the upper extremities and increases extension in the lower extremities. When a patient is lying supine, there will be greater flexor tone in the upper extremities and greater extensor tone in the lower extremities than when he is lying prone. This influence of the static labyrinthine reflexes on muscular tone makes positioning of the patient with an upper motor neuron lesion an important factor during therapeutic exercise, as well as during rehabilitation training. If spasticity in finger and wrist flexors interferes with muscle reeducation of the hand, greater relaxation can be obtained by placing the patient in the prone and head down position.

The kinetic labyrinthine reflexes are initiated by stimulation of receptors located in the semicircular canals of the inner ear by rotation of the head. As the head rotates, there will be increased flexion in the upper and lower extremities and in the muscles of the trunk on the side toward which the patient is turning and an increased extension on the side away from which the patient is turning. This reflex response has been described by Fay as the salamander crawling pattern.[4]

Another supraspinal reflex of importance in maintaining upright posture is the positive supporting reaction.[5] This is an augmentation of the extensor thrust reaction described previously under the spinal reflex patterns. Stimulation by pressure on the ball of the foot to stretch the triceps surae and stretching of the flexors of the toes will cause an extension of the lower extremity at the hip, knee, and ankle, with co-contraction of flexors and extensors to convert the extremity into a solid pillar for support when walking. Conversely, a negative supporting reaction is obtained by vigorous passive flexion of the toes, in response to which there is relaxation of the extensor muscles of the ankle, knee, and hip, and contraction of the flexors of those joints produces a withdrawal response. Alternate evocation of the positive supporting reaction and the negative supporting reaction will reflexly produce flexion and extension of the lower extremity in an apparent walking pattern which is independent of volitional control. This reflex activation of the lower extremity is frequently utilized when carrying out range-of-motion exercise of the lower extremities of patients who have severe spasticity in order to decrease the active muscular resistance against which the therapist must work. These reflexes are also utilized at times to facilitate volitional reciprocal flexion and extension of the lower extremities to develop the habit patterns for walking.

RIGHTING REFLEXES

A further group of supraspinal reflexes of importance in posture are the righting reflexes which assist the body in assuming and maintaining the upright posture (Table 3). The first of these is the optical righting reaction which requires an intact pathway from the retina to the occipital cortex and intact subcortical pathways. In response to visual stimuli, the head is righted so that it is oriented in an upright position in relation to the environment. For utilization of this reaction, the environment must be familiar. Psychological studies have shown that when trick rooms are used to produce the optical illusion that verticality is not in the direction of the pull of gravity, the subject may become confused and hold the head in a position comparable to the

TABLE 3

RIGHTING REFLEXES INVOLVED IN BALANCE

Optical righting reaction
Labyrinthine righting reflex on the head
Neck righting reflex of head on the body
Body righting reflex on the head
Body righting reflex on the body

apparent vertical position of the room and, consequently, not upright in relation to gravity. Damage to the nervous system which produces a distortion of visual perception may produce this same abnormal orientation.

The second righting reflex is the labyrinthine righting reflex on the head. The stimulus arises from the utricle in the inner ear. The reflex center is in the midbrain, lower than that of the optical righting reflex. The equal effect of gravity on the otoliths in the utricles bilaterally causes the body to hold the head in a vertical position. If one of the labyrinths or its pathway is destroyed, the head will be tilted toward the damaged side. For example, the hemiparetic patient with damage of one labyrinthine pathway will lean toward the paretic side which will seriously interfere with bodily support. Such patients have great difficulty because, reflexly, they fall toward the paretic side.

A third righting reflex describes the effect of the position of the neck on the orientation of the trunk. When the head is held in an upright position, if the neck is flexed, bent laterally, or rotated, through a reflex centered in the midbrain, there will be contraction of muscles to orient the body in a linear relationship to the head so that the neck is straightened. This is the so-called neck righting reflex of the head on the body.

There is also a body righting reflex that acts on the head which is centered in the midbrain. If the body is lying on one side and there are no labyrinthine and optical righting reflexes, the sensory pressure due to contact with the skin on that side causes contraction of muscles to raise the head to the upright position. This reflex can be neutralized by applying equilateral pressure on the two sides of the body. Thus, although an animal is lying on one side, the head will not be lifted.

Finally, the righting reflex of the body on the body has its center in the medulla. Pressure on one side of the body causes orientation of the posterior part of the body in an upright position. As a result of unilateral pressure, first the sacral end of the body is lifted, then the lumbar section is righted, and, finally, the anterior torso is oriented in the upright or ventral lying position.

It may be seen that these reflex controls of posture are multiple; some may augment while others oppose a given posture. Because of this multiplicity of reflex controls, a certain amount of compensation is possible when there is partial damage to the central nervous system. Often the compensation is incomplete, or a prolonged period of time may be required for the body to make an adjustment.

The problems which are frequently seen in the patient with hemiplegia are typical of some of these adjustments. If there is hemianesthesia in the hemiplegic patient with a resulting decrease of sensory input and decreased activity of the spinal cord on the paretic side, there is more severe paresis and more difficulty in controlling the hemiplegic side. In some cases, muscular responses on the paretic side of the body may be increased by increasing sensory stimuli to that side. When there are painful stimuli, such as those resulting from stretching of contractures, irritation due to bladder infection, or irritation from decubiti, flexion reflexes or mass spinal reflexes may be initiated, interfering with extension of the lower extremities for support in standing or walking. Patients who have this type of reflex activity have great difficulty in learning to stand or walk until the flexion reflexes are diminished by reducing the noxious sensory stimuli. The patient who has tightness or spasticity of the triceps surae has an increased extensor thrust reaction every time he stands on his toes, and he has difficulty both in getting his heel to the floor and in flexing the hip and the knee to lift the foot from the floor. Inhibition of the positive supporting reaction by bracing the ankle or by procedures which decrease the sensitivity of this reflex increase the ability to flex the hip and knee during ambulation.

This brief review of reflexes involved in balance emphasizes the need for a thorough understanding of the neurophysiology of balance in order to adapt therapeutic procedures to the disability of the patient and help him to regain optimal muscular control for balance and for walking.

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History of Physical Medicine at the University of Minnesota from 1945 to 1965

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THE TWENTY YEAR span from 1945 to 1965 was a period of remarkable growth and development in physical medicine. The necessary talent, coupled with the motivation for teaching and training doctors in the proposed new specialty of physical medicine, for teaching and training occupational therapists and physical therapists, and for conducting research in basic sciences as well as on clinical problems was available in the medical school of the University of Minnesota at the time when our country became aware of the needs for and potentialities of physical medicine.

During the civilian manpower shortage which developed when men were drafted for service in World War II, industry was forced to hire some handicapped people in order to meet production schedules. When the performance of these people was evaluated it was found that their productivity was good—absenteeism and accidents were actually less among this group than among the nonhandicapped workers and they tended to stay at their jobs.

At the end of World War II there were many disabled soldiers who required rehabilitation before they could return to civilian duties. The impetus to study the needs for physical medicine on a nationwide basis was given financial support by Bernard M. Baruch, financier, philanthropist, and advisor to presidents, whose father, Dr. Simon Baruch (1840-1921), was a physician practicing medicine during the Civil War. Dr. Baruch had been convinced of the efficacy of physical agents, and in particular, of the value of hydrotherapy for the treatment of disabilities. In memory of his father, Bernard Baruch made available an initial sum of 1.1 million dollars of his personal fortune to be used to determine the needs of the people of our country for physical medicine.

A committee of distinguished doctors and scientists from educational institutions and the armed services was selected in 1943 to promote the advancement of the science of physical medicine. Dr. Frank H. Krusen,* a state and national leader in the development of physical medicine, became the director of the work of the Baruch committee. A committee on war and postwar physical rehabilitation and reconditioning was also established in the same year. The first report of the committee stressed the need for a comprehensive program, including physical rehabilitation, psychosocial treatment, and vocational retraining for demobilized veterans, as well as for those members of the civilian population who each year become handicapped by disease and accidents or have congenital handicaps. The number of physicians qualified to teach and use physical medicine was found to be extremely limited so the recruitment and training of new men had to be accelerated. It was realized that a great deal more basic and clinical research would have to be done to build up the body of knowledge in the discipline of physical medicine.

Included in this first report was a statement which John Galsworthy had made in 1919 at the Allied Conference on the aftercare of disabled men, held in Washington, D.C.:

> Restoration is at least as much a matter of spirit as of body and must have as its central truth: Body and spirit are inextricably conjoined. To heal the one without the other is impossible. If a man's mind, courage, and interest be enlisted in the cause of his own salvation, healing goes on apace, the sufferer is re-made; if not, no mere surgical wonders, no careful nursing, will avail to make a man of him again. Therefore I would say: "From the moment he enters the hospital, look after his mind and his will; give him food; nourish him in subtle ways; increase that nourishment as his strength increases. Give him interest in his future. Light a star for him to fix his eyes on, so that, when he steps out of the hospital, you shall not have to begin to train one who for months, perhaps years, has been living, mindless and will-less, the life of a half-dead creature."
>
> That this is a hard task none who knows hospital life can doubt. That it needs special qualities and special effort, quite other than the average range of hospital de-

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votion, is obvious. But it saves time in the end, and without it success is more than doubtful. The crucial period is the time spent in the hospital. Use that period to recreate not only the body, but mind and will power, and all shall come out right; neglect to use it thus and the heart of many a sufferer and of many a would-be healer will break from sheer discouragement. A niche of usefulness and self-respect exists for every man however handicapped; but that niche must be found for him. To carry the process of restoration to a point short of this is to leave the cathedral without spire. To restore him and with him the future of our countries, that is the sacred work.

It was felt that since physical medicine encompassed such a broad field, these terms required definition: physical medicine, rehabilitation, and occupational therapy. The following definitions were evolved: (1) Physical medicine includes the employment of the physical and other effective properties of light, heat, cold, water, electricity, massage, manipulation, exercise, and mechanical devices for physical and occupational therapy in the diagnosis or treatment of disease. (2) Medical rehabilitation is the restoration of people handicapped by disease, injury, or malformation as nearly as possible to a normal physical and mental state. Medical rehabilitation fills the gap between the customary end point of medical attention and the real necessities of many patients. (3) Occupational therapy provides a graded program of activity to restore maximal physical and mental function or seeks to divert a person and improve morale by arousing his interest, courage, and confidence.

Speaking of the establishment of combined programs of teaching and research in the medical schools, the Baruch committee recommended that there be 2 key men in physical medicine—one, a physician, with graduate training in physical medicine and the other a research man specially trained in physics or physiology, or both. The committee further recommended that "These 2 key men should gather around them a group of interested teachers and research workers in allied fields and should have working with them a number of fellows who will eventually be trained to teach either the clinical aspects or the basic research aspects of physical medicine in other medical centers."

Following its preliminary survey, the committee recommended that the funds which had been made available by Mr. Baruch be distributed as follows: Three large grants to centers to become "model" centers for the country and smaller amounts to educational institutions to be used for the purpose of establishing or strengthening existing programs of teaching and research in physical medicine. Columbia University received $400,000 for the development of a ten-year program in physical medicine, New York University received $250,000 to develop a center in physical medicine, with special emphasis to be placed on structural mechanics of the body, and the medical college of Virginia received $250,000 for a center of physical medicine with special emphasis on hydrology.

The University of Minnesota Medical School was one of the centers chosen to receive support from the Baruch Foundation and an expanded program of teaching and research in the field of physical medicine was begun in January 1945 with the receipt of $40,000 for this purpose.

A committee was formed to direct the project at the university. This committee, under the chairmanship of Dr. M. B. Visscher, professor and head of the department of physiology, consisted of Dr. Harold S. Diehl, dean of the medical sciences; Dr. Miland E. Knapp, director, division of physical therapy; Dr. Frank H. Krusen, professor of physical medicine, Mayo Foundation; Dr. William A. O'Brien, director of postgraduate medical education; Dr. Wesley W. Spink, associate professor of medicine; and Dr. Karl W. Stenstrom, professor of biophysics.

Men who were to become "the 2 key men" were well along in their training. Dr. Frederic J. Kottke, M.D., who had a Ph.D. in physiology, with a minor in pathology, was chosen to be a Baruch Fellow in physical medicine. Dr. W. G. Kubicek was completing his advanced studies in physiology and physical chemistry and carrying out basic research in physiology.

In the report to the committee for the period ending Dec. 31, 1946, Dean Diehl stated in conclusion: "The Minnesota committee for the administration of the Baruch grant is now fairly well satisfied with the general plans for work in physical medicine at the University of Minnesota. Until recently it was impossible to obtain many competent students and workers in this field. We now have nearly completed the training of Dr. F. J. Kottke, who will soon, we expect, take over responsibility for work in this field and we have several other competent workers in training.

"During the past year, we expended $9,400 of Baruch funds and we expect to spend somewhat more during the current years.

"We propose to 'make progress slowly' by basic research on fundamental problems, because we have no hope of being able to solve the practical problems of physical medicine without more fundamental research. This is not the spectacular way to proceed, but we are sure that it is the only way to be certain of ultimate success."

On July 1, 1947, Dr. Kottke was made assistant professor of physical medicine. Dr. William G. Kubicek, assistant professor of physiology, became a permanent member of the faculty of physical medicine in 1948. The departmental office was located on the second floor of a wooden temporary building west of the University Hospitals. Occupational therapy had 2 small rooms on the first floor of this building, while the physical therapy clinic had the physical facilities in University Hospitals alluded to by Dr. Knapp. Research was carried on in a laboratory known as West 200 of the hospital and in one of the physiology laboratories on the fourth floor of Millard Hall.

In addition to Drs. Knapp, Kottke, and Kubicek, the teaching staff of the division of physical medicine listed in the Baruch report for the period of Jan. 1, 1948 to June 30, 1949 was composed of: 1 clinical instructor, 3 fellows in physical medicine, 1 director of the school of physical therapy, 1 instructor in the school of physical therapy, 1 director of the school of occupational therapy, 1 instructor in the school of occupational therapy, and 1 instructor of biophysics.

In the clinic at that time, there were 1 physical therapy supervisor, 4 physical therapists, and 1 fever therapist, while in the research section there were 2 medical technologists.

YEARS OF EXPANSION AND DEVELOPMENT

Financial support from the Baruch Foundation ended in 1951, but a substantial beginning had been made. Under the energetic leadership of Dr. Frederic J. Kottke, augmented by the strong support of Dr. Kubicek in the area of research, the years from 1947 on were devoted to the expansion of physical facilities at the University of Minnesota to permit total treatment and care of patients requiring rehabilitation services and to the increase in scope of research related to problems encountered in physical medicine. These years were also devoted to the strengthening of the curriculum in physical medicine. Physical medicine was a division of the department of radiology until 1952. In 1952 physical medicine and rehabilitation was made an autonomous department of the medical school with Dr. Kottke as head.

The first major expansion was the location of the rehabilitation center on the 7th and 8th floors of the Mayo Memorial. The center was activated in 1954. Only the persons directly involved can truly appreciate the amount of planning and coordination which brought it about. Civic groups, including The Mayor's Committee for the Employment of the Handicapped during the time when Hubert H. Humphrey was mayor of Minneapolis, and Crippled Child Relief, under the leadership of Mrs. Della Olsen, provided strong support for its establishment in Minnesota. Crippled Child Relief initially made a gift of $58,000 toward equipping the center. The Minnesota legislature appropriated funds for the construction of the facility. In 1961 Dr. Glenn Gullickson, Jr., was made director.

When completed in 1954, the new rehabilitation center was lacking space for speech therapy, a sufficient number of examining rooms, and space for a prevocational try-out shop for the adequate evaluation of the employment potential of patients. Funds from the state division of Vocational Rehabilitation, the national office of Vocational Rehabilitation, and Crippled Child Relief made possible an expansion on the south and southwest wings of the 7th floor of the Mayo Building. Many persons worked for this expansion. Governor Orville Freeman was actively interested in the potentials of this project and encouraged its support. These facilities for speech therapy, work evaluation, vocational counseling, and medical examination were activated in 1957.

Occupational therapy required laboratory space so that students could become experienced in the handling of power equipment. Crippled Child Relief again followed its policy of assisting the rehabilitation program by providing $10,000 toward the remodeling of part of the former physical therapy clinic on the first floor of the University Hospitals to make a modern laboratory and classroom. Students began using the laboratory in 1958.

Treatment of patients, research, and teaching continued at an accelerated pace in the new facilities. Yearly reports from the department attested to growth in all areas.

Several factors were operating which would ultimately lead to the construction of a separate facility for children. The number of children who could be treated was limited, among other things, by available ward facilities for long-term patients and by lack of treatment space especially designed and equipped for children in the center itself. The hospital ward occupied by adults (Station 22) was needed for expansion of the organ transplant service. Dr. Kottke realized that a separate children's center which would not only provide a planned rehabilitation station for the adults from Station 22 but also a rehabilitation station and special treatment areas for 20 children, as well as expanded laboratory facilities and classrooms, would enable the staff to do a better job and permit a greater opportunity

for fellows in physical medicine, therapy students, and others being trained in various phases of rehabilitation.

Exploration of possible sources of funds was begun in 1960. Encouragement and assistance were provided by Dr. Robert N. Barr and Dr. Helen L. Knudsen of the Minnesota Department of Health. By January 1962, it was announced through various press releases that there would be a children's rehabilitation center constructed. Funds for the building came from a number of sources. Proceeds from the sale of the William Eustis property provided funds to cover 55% of the cost and Hill-Burton funds and private sources provided the remainder. The 6½ floor Children's Rehabilitation Center was activated in the summer of 1964 and dedicated on Nov. 7, 1964—ten years after the opening of the rehabilitation center in the Mayo Building and five days after the election of Hubert H. Humphrey to the office of vice-president of the United States.

With the completion of the center, a need which had been indicated in a report by Dr. Alice K. Brill (a Baruch Fellow in physical medicine, who had been doing research in pediatrics) to the University of Minnesota Hospitals staff meeting on May 11, 1945, had been filled. The title of Dr. Brill's presentation was "The Need of Physical Therapy in Pediatrics."

Physical Medicine and Rehabilitation* provides a three-year residency which satisfies the requirements for formal training established by the American Board of Physical Medicine and Rehabilitation. Programs leading to the M.S. and the Ph.D. degrees are also available for doctors who aspire to the teaching of physical medicine and rehabilitation. Summer fellowships are offered to medical students. Under the comprehensive clinic plan, undergraduate doctors have an opportunity to follow patients in rehabilitation. Drs. Kottke, Kubicek, Gullickson, and Knapp direct the programs of advanced study in the field.

The occupational therapy course became established as a four-year degree course in 1946 and received accreditation in 1948. Miss Borghild Hansen was director of the course from its beginning to her death in 1966. The physical therapy course became a four-year degree course in 1948. Ruby Green (Overmann) became the director of the course in 1947 and was succeeded by Wilbur Moen in 1957.

By 1965 the teaching staff, besides Drs. Kottke, Kubicek, and Knapp, had increased in number to 45.

In the clinic the number of physical therapists at various levels of responsibility totaled 18 and occupational therapists numbered 16. Full-time laboratory personnel in research numbered 5 as well as a kinematics mechanician and an electronics mechanic.

The staff also included speech pathologists, a social worker, vocational counselors, and psychometrists.

In 1961 the Vocational Rehabilitation Administration designated the University of Minnesota Medical School and Sister Kenny Rehabilitation Institute to be 1 of 5 regional rehabilitation centers to receive financial support for teaching and research in the field of rehabilitation.

With this support the rehabilitation center will be able to continue improving the curriculum in physical medicine and rehabilitation and thereby be enabled to render better service to the handicapped, not only in Minnesota but nationally and internationally through the people who have been trained at the center. The department of physical medicine and rehabilitation of the medical school of the University of Minnesota will thus continue working toward the more complete realization of the words carved on Northrup Memorial Auditorium:

"Founded in the faith that men are ennobled by understanding. Dedicated to the advancement of learning and the search for truth. Devoted to the instruction of youth and the welfare of the state."

*In 1953 the term "rehabilitation" was officially added to the designation of the specialty of physical medicine.

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The Relationship of the Staff Physician to the Department of Physical Medicine and Rehabilitation of a Community Hospital

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THE RELATIONSHIP of the staff physician to the department of physical medicine and rehabilitation in a community hospital can be considered from three separate approaches, depending upon the administrative situation in the department and the presence or absence of a physiatrist to direct the department and conduct consultations.

FULL-TIME PHYSIATRIST IN CHARGE

When a full-time physiatrist is in charge of the department, the responsibility of the staff physician toward the department is much less demanding than it would be if a part-time physiatrist were in charge or if no physiatrist were available. When a physiatrist devotes all his time to the department, the responsibility of the staff physician relates primarily to [1] selection of patients who need physical treatment and [2] referral of these patients to the department with adequate information as to the history, diagnosis, and pertinent physical findings, as well as warnings about the dangers of physical or psychologic damage which might occur when treatment is given.

The exact responsibilities of the staff physician will depend upon the administrative policies of the department. In some instances the referring physician is expected to furnish diagnostic information but is not expected to participate in the prescription of treatment, since the physiatrist would write the orders for specific treatment. Usually, in this circumstance, the best method is direct referral of the patient to the physiatrist, who then takes a complete history, conducts an examination, confirms the diagnosis or makes a new one, and prescribes the treatment. Even when this sort of arrangement prevails, it is often advisable for the referring physician to suggest a mode of treatment, although it is not necessary for him to give specific orders.

In fact, in many departments, even when a full-time physiatrist is in charge, arrangements can be made so that the referring physician, if he wishes, may prescribe the treatment himself. When this type of arrangement is in force, the staff physician has the entire responsibility for the diagnosis and treatment, while the physiatrist's responsibility is limited to making certain that the treatment ordered is not harmful, that it will be helpful to the patient, and that it is carried out according to the orders by accepted methods and techniques. In my opinion, it is necessary for the physiatrist to see the patient, to confirm the history, to approve the results of examination and the diagnosis, and to concur in the prescription of treatment if this type of supervision is to be his responsibility.

Some physiatrists do not agree with this point of view. They believe that supervision of treatment can be done adequately without seeing the patient at all. The legal responsibilities of the prescribing physician and of the supervising physiatrist enter into this problem but are not yet well defined or understood.

Certainly, should a difference of opinion arise between the physiatrist and the referring physician, they should always confer with each other and arrive at a mutual agreement before any changes in the diagnosis or orders are made.

PART-TIME PHYSIATRIST AVAILABLE

When a part-time physiatrist has charge of the department, the responsibility of the staff phy-

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sician is the same as the foregoing in respect to selection and referral of patients. However, if it should happen that the physiatrist is not immediately available and treatment is needed at once, the staff physician should give orders which are as specific as possible. He should ascertain later that the physiatrist examined the patient and prescribed treatment in detail. In turn, the physiatrist has the responsibility of consulting with the staff physician on request not only because the staff physician was the one who referred the patient for treatment but also because of the mutual benefits of informal discussion. In this way the physiatrist can acquaint the staff physician with physical methods and stimulate his recognition of the benefits of physical medicine in his practice.

NO PHYSIATRIST IN CHARGE

When no physiatrist is available for consultation, the responsibility of the staff physician becomes much greater. Not only must he select the proper patients to refer for treatment but also he must write a complete and adequate prescription. This prescription should include the specific modalities to be used, duration of treatment, dosage, and frequency. He should also warn the therapist specifically about possible dangers inherent either in the treatment or in the patient. If the staff physician is not sufficiently acquainted with the modalities used to be able to prescribe them intelligently, he should confer with the therapist, explaining what he desires to do and the pathologic problems present. The therapist can then give suggestions as to what types of treatment would be suitable. However, the prescription never should be made by the therapist; it must be made by the physician.

Legally, diagnosis and prescription of treatment are strictly confined to the province of the physician and should never be delegated to a person who is not a physician. The physician who gives the orders ultimately will be responsible if untoward incidents develop or a malpractice suit results. This is true even if the therapist actually makes the prescription, because the physician, by the implications of his license to practice medicine, is the one who must be in charge of the patient and the treatment. In an incident such as the one just mentioned, the mere fact that the physician did not write the prescription could be represented as evidence of his negligence.

EXPLICIT DIRECTIONS FOR TREATMENT

The basic principles of the prescription of physical procedures require that the prescription be as nearly accurate as is the prescription of drugs. Too frequently a patient is sent to a therapist with a prescription which reads "physical therapy" or even worse, "physiotherapy." Such a prescription is equivalent to sending the patient to a druggist with a prescription reading simply "medicine." Any physician who would issue the latter prescription would soon be the object of critical attention from his medical society. Yet, in many instances, highly respected physicians refer patients for "physical therapy" or "physiotherapy" and think nothing of it.

This type of prescription is a confession of ignorance on the part of the physician, since he usually does not know what to order. The hiatus in knowledge may be traced directly to the lack of instruction in physical medicine in medical schools throughout this country. It is a fact that only about half of the medical schools in the United States have departments of physical medicine headed by physiatrists to teach students the basic principles of this specialty. In general, when this type of instruction is offered at all, the time allotted to it is even less than the time allowed for other subjects.

Moreover, it is not sufficient to give a general prescription, such as "heat," "massage," and "motion," leaving the details to the discretion of the therapist.

If heat is prescribed, the exact type of heat to be used should be specified. This might be radiant heat in the form of infrared radiation from a dark body radiator, a carbon-filament bulb, or a tungsten-filament bulb. The wattage to be used, the distance of the lamp from the patient, and the duration should all be specified.

Conductive heat might be used in the form of hot packs, hydrocollator packs, an electric pad, an aqua K pad, or a whirlpool or Hubbard tank. In this case, the temperature and duration of treatment should be specified.

Conversive heat might be ordered. This is generated by shortwave diathermy, microthermy, or ultrasound. In the first two instances, heat is usually ordered to comfortable tolerance and the duration is specified. In the latter instance, the treatment is prescribed in terms of watts per square centimeter of the transducer head and the duration is specified.

The heat is then followed by massage. I usually find it advisable to specify the sequence of treatment, meaning that the massage should follow the heat. The type of massage also should be specified. Examples are deep sedative massage, superficial massage, fibrositic massage, and so on. Generally, I think it is best not to specify the duration of the massage, because the thera-

pist can determine better than I the effect the treatment is having on the patient and will discontinue it when the desired results have been obtained, obviating overtiring or irritating the patient from unduly prolonged treatment.

The type of motion or exercise should also be specified, for instance, range-of-motion exercises, active-assisted exercise, or resistive exercise of a type denoted, such as DeLorme's heavy-resistance exercise technique or isometric exercise. Here again, the duration of exercise as a rule is not specified, because it will depend upon the tolerance of the patient and it will vary from day to day.

The specific areas to be treated should be included in the prescription, and the frequency of treatment should be stated, such as whether it should be daily, every other day, twice a day, and so forth. A prescription thus might read as follows: "For physical therapy; area to be treated: left shoulder. First, shortwave diathermy to comfortable tolerance for 20 minutes, followed by deep sedative massage, followed by scapulohumeral stretch, followed by active exercise on the shoulder wheel designed to increase range of motion. The treatment is to be given daily. Warning: Do not hyperflex or hyperabduct the shoulder because of danger of redislocation."

When occupational therapy is to be prescribed we cannot be so specific about the type of treatment. This depends upon the interest of the patient and, in most instances, upon the inventive gifts of the therapist in adapting equipment to accomplish the desired results. However, the purposes of the treatment should be specified and the duration and the general methods or projects should be set down in as much detail as possible. Again, it is important that the therapist be given the required warnings or suggestions about the treatment which seem pertinent.

For instance, occupational therapy for the same patient as in the previous prescription might be ordered as follows: "Occupational therapy to increase range of motion of the shoulder and strengthen the abductors. Suggest the use of the large loom, but this may be modified to suit the patient's desires. Duration to patient's tolerance, but no longer than one-half hour. Frequency: twice daily. Warning: Do not hyperflex or hyperabduct the shoulder because of danger of redislocation."

COMMENT

For the past three years I have been visiting hospitals and rehabilitation centers in outlying portions of Minnesota where at least physical therapy and sometimes occupational therapy are available to the practicing physician. Well-trained therapists manage the departments, but no physiatrist is available in these communities.

During monthly visits, clinics, lectures, and consultations are held in an attempt to bring some degree of postgraduate education in physical medicine and rehabilitation to physicians in their own communities, with their own patients as teaching material. These physicians are almost too willing to admit their lack of knowledge of physical medicine. They tend to depend too much upon the therapist for advice which he really is not qualified to give. This is disturbing to the therapist as well as to the physiatrist.

Many of these general practitioners are eager to learn more about physical measures which will benefit their patients. The visits I have mentioned constitute one way of providing continuing education in a field which is poorly understood by physicians generally.

End of Symposium—Physical Medicine and Rehabilitation

Two-Chambered Right Ventricle Associated with Primary Pulmonary Hypertension

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THE DIVISION of the right ventricle into two chambers by an aberrant hypertrophied muscular band is a relatively rare congenital malformation. In this report, a case of two-chambered right ventricle associated with primary pulmonary hypertension is presented.

CASE REPORT

A 20-year-old Hindu primipara who delivered a full-term breech baby at King Edward VII Memorial Hospital on July 18, 1963, suddenly developed substernal chest pain with breathlessness and sweating on the fourth postnatal day. Her past history did not record significant illness of any kind.

On examination, the patient was found to be breathless, cold, and clammy. She had pulse rate of 130 per minute, respiratory rate of 46 per minute, blood pressure of 90/60 mm. Hg, and raised jugular venous pressure. There was no cyanosis or clubbing. Examination of the cardiovascular system revealed tachycardia, right ventricular gallop, and loud split pulmonary second sound. A short systolic ejection murmur was audible in the pulmonary area. Examination of the abdomen revealed a palpable, tender liver at 2 fingers. There were crepitations at the lung bases.

The patient was treated with sedatives, digitalis, and anticoagulants. Her electrocardiogram revealed evidence of right ventricular enlargement and abnormal ST-T changes. The skiagram of the chest showed minimal cardiac enlargement with prominently bulging pulmonary conus and pulmonary plethora (Fig. 1).

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She continued to have occasional attacks of breathlessness and palpitations which were treated with injections of aminophylline. On the tenth postnatal day, she developed sudden severe breathlessness and expired. Autopsy revealed the following interesting features.

Gross. The heart weighed 298 gm. and was slightly enlarged, mainly due to right ventricular hypertrophy. The visceral pericardium was smooth. Just below the pulmonary ring, a small dilatation was present in the outflow tract (seen on posterioanterior view of the chest as prominently bulging pulmonary conus [Fig. 1]), indicating constriction proximally. On opening the heart, the right atrium was enlarged in size and revealed smooth endocardium (Fig. 2A). A



Fig. 1. Skiagram of chest showing prominent pulmonary conus



Fig. 2A *(left)* First chamber of the right ventricle. Arrow points to the opening which leads to the second chamber. B. *(right)* Second chamber of the right ventricle with pulmonary valve. Arrow points to the communication between the two chambers.

thrombus was seen in the right atrial appendage. The tricuspid valve opened into a chamber a little smaller than the usual right ventricle. The myocardium of this chamber was markedly hypertrophied and the endocardium smooth. Instead of the usual outflow tract, a small circular opening 2 cm. in diameter was seen in the muscular septum which led into another small triangular chamber (Fig. 2B), the base of which was formed by the pulmonary ring. It measured 5 cm. from base to apex. The thickness of the myocardium was the same as that of the first ventricle. It was communicating only with the pulmonary artery. There was no communication between either of the chambers and the left ventricle. The pulmonary ring and cusps were normal, and the pulmonary artery was larger in size and showed few patches of atheroma. No abnormality was seen in the left atrium, mitral valve, and left ventricle.

Both the lungs were normal in size and shape. Pleural surfaces were smooth and shiny; the consistency was subcrepitant all over. The pulmonary arterial tree, after dissecting, did not show any embolus, and a few patches of early atheroma were seen in the intima of main branches. The cut surface of the lung tissue showed normal appearance, with no evidence of consolidation or infarction.

Histology. Lungs. The pleura and lobular septa were normal, alveoli were clear and thin walled, and alveolar capillaries were inconspicuous. The arterioles were normal, but the small-sized muscular arteries showed remarkable change, most of them with marked narrowing of lumina due to proliferative intimal thickening. A few of them were completely obliterated by thrombus (Fig. 3). Recanalization of thrombus was seen in an occasional vessel. Large-sized elastic arteries showed atheromatous patches in the intima. No significant histological change in the heart was seen.

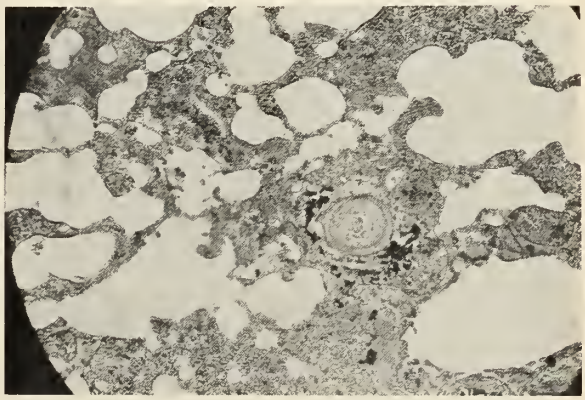


Fig. 3. Microphotograph of lung showing a medium-sized muscular artery with obliterative changes

DISCUSSION

The occurrence of cor triventriculare due to two-chambered right ventricle is itself a rarity, though a few cases have been described. Though association of this anomaly with ventricular septal defect and pulmonary stenosis has been described, that of primary pulmonary hypertension is not recorded.

Keith,[1] who first described 19 cases in 1909, considered this anomaly a result of incomplete absorption of bulbus cordis into the right ventricle. Grant and associates,[2] in their illuminating study of the architecture of the right ventricular outflow tract, have described this

anomaly as a result of hypertrophy of the oblique component (cf. bulbar musculature), probably as a result of injury to prediffercntial tissue.

Hartmann and co-workers[3] reported a clinical study of 9 cases over a five-year period. Clinical findings in all cases resembled those of acyanotic Fallot's-Harsh systolic murmur, best heard at the left sternal border with a palpable systolic thrill. Pulmonary second sound was diminished, and the electrocardiogram was normal in 5 cases. Others showed varying degrees of right ventricular hypertrophy or incomplete right bundle-branch block. Chest X-ray revealed evidence of right ventricular enlargement. Pulmonary vasculature appearance varied from patient to patient, depending on the degree of left to right shunt. Left ventricular enlargement was described in 5 cases with left to right shunt. No case had any evidence of pulmonary hypertension. Kinare and Sen[4] have reported a case of cor triventriculare due to two-chambered right ventricle of sizable degree associated with fenestrated cusp.

The present case is perhaps of greater interest due to its associated primary pulmonary hypertension. The third ventricular chamber was big enough to be seen as dilatation in the outflow tract, misinterpreted on the skiagram of the chest as enlarged pulmonary conus.

Review of literature on primary pulmonary hypertension records over 80 cases. Donald Health[5] in his report of 4 cases describes the pathological changes in pulmonary vasculature diagnostic of this condition. Evans and associates[6] and Paul N. Yu[7] describe dyspnea, fatigue, palpitations, and attacks of syncope as the presenting manifestation of primary pulmonary hypertension. Sometimes dyspnea could be quite abrupt and developed unexpectedly during exercise or after trivial illness. They reported 2 deaths during pregnancy due to sudden dyspnea and syncope. Cardiac pain usually accompanies dyspnea. Cutler and co-workers[8] have described a case of primary pulmonary hypertension in association with aortopulmonary fenestration.

In the present case, obliterative changes of pulmonary hypertension were seen in the pulmonary vasculature, but no other lesion accountable for the same was detected in the heart. No clinical evidence of thromboembolic phenomenon was available from the past history. The degree and duration of histological changes observed in the lungs were sufficient to produce atheroma in the main pulmonary arteries and could not have been a consequence of the patient's recent pregnancy.

SUMMARY

A case of two-chambered right ventricle associated with primary pulmonary hypertension has been described, including autopsy findings.

We are grateful to the dean, K.E.M. Hospital, Bombay, for permitting us to use the hospital case records and to publish this case.

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Therapy of Hemangiopericytoma with Pulmonary Metastasis

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HEMANGIOPERICYTOMA is a rare vascular tumor which has been accepted as a separate pathological entity. Stout and Murray[1] are credited with the original description in 1942. The hemangiopericytoma belongs to a group of exasperating and peculiar tumors, occasionally benign, but usually malignant, always of unpredictable behavior.

The anatomical distribution is limitless since this type of tumor can arise in any tissue where capillaries exist.

Accumulated experience indicates that there is no particular sex, race, or age group distribution.

Cases have been reported[2] in which recurrence occurred twenty-six years following the original surgical excision, and others which demonstrated a rapid clinical course with massive generalized metastasis.[3] Out of a series of 32 malignant hemangiopericytomas, Dr. A. P. Stout[1] found 5 that originated in the upper extremities; about half of the 32 metastasized.

Grossly, the tumor is more or less circumscribed but poorly encapsulated and frequently locally inactive. On microscopic sections it consists of capillaries surrounded by many layers of round or spindle shaped contractile cells known as pericytes, recognized about forty years ago by the histologist Zimmerman. The neoplastic cells lie outside the vascular sheath. It is difficult to predict the course by the histological picture. Lymphatic, or more usually, hematogenous metastasis can occur at any time.

Radical extirpation of the lesion by surgery is considered the treatment of choice. In more superficial lesions without any evidence of local invasiveness, wide local excision may be the acceptable method of therapy; should local recurrence occur, however, radical surgery is by all means indicated.

Radiotherapy as an adjunct to surgery, seems to be of limited usefulness.[4] Its real value has not yet been well established. Its value is doubtful in the treatment of this particular type of tumor. Experience with chemotherapy is limited. The use of mechlorethamine (Mustargen®) and

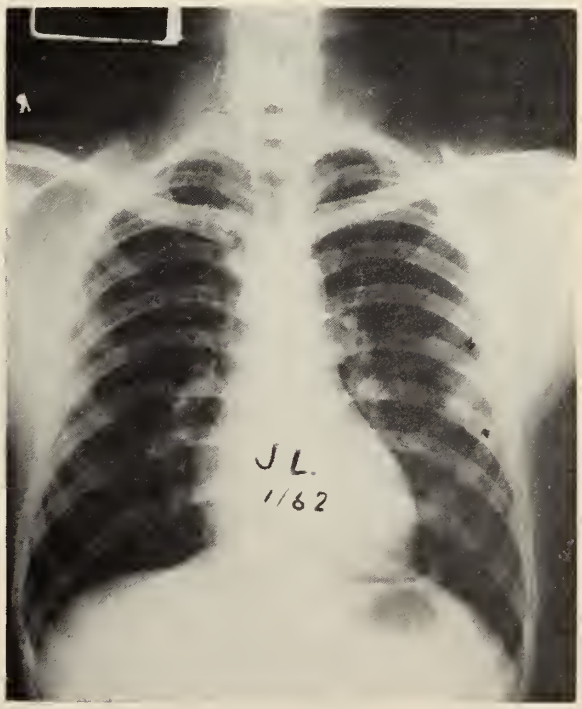


Fig. 1. Nodular density in left mid lung asymptomatic

WILLIAM BOGEDAIN *and* JOHN CARPATHIOS *are from the Department of Thoracic Surgery, Mercy and Timken-Mercy Hospitals, Canton.*

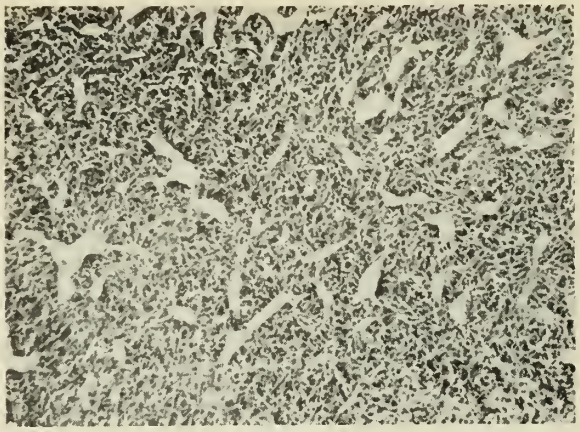


Fig. 2. H and E × 90. This area shows a differentiated portion of the tumor, with endothelial-lined sinusoids separated by cords of perithelial tumor cells.

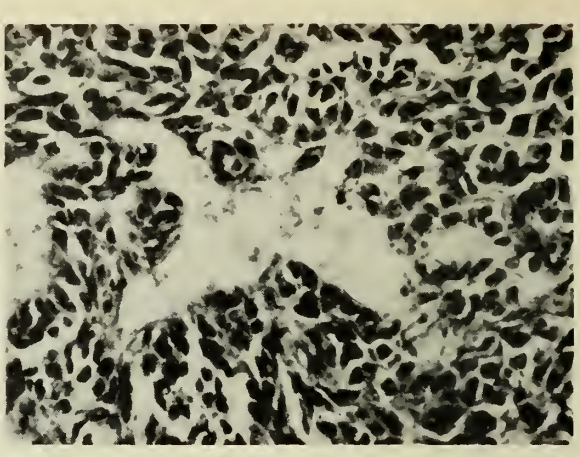


Fig. 3. H and E × 350. Higher power view of the vascular sinusoids containing red blood cells, separated by fusiform perithelial cells

cyclophosphamide (Cytoxan®) in this case was of doubtful benefit in the treatment of this tumor.

CASE REPORT

A 16-year-old boy was admitted to Mercy Hospital in July 1959, with an asymptomatic, round, fast growing mass in the soft tissues of the right forearm.

Surgical removal of this tumor was carried out. The site of the lesion in the soft tissues, the encapsulation, the clinical progress, the microscopic appearance, and reticular structure helped to establish the pathological diagnosis of malignant hemangiopericytoma.

In May 1960, the patient was readmitted to the hospital with recurrence in the upper portion of the scar. Radical amputation was advised but strongly rejected by the patient. Wide excision combined with axillary node dissection was carried out.

In January 1962, the patient was readmitted with an asymptomatic nodular density in the left mid-lung field (Fig. 1).

The lesion was removed by segmental resec-

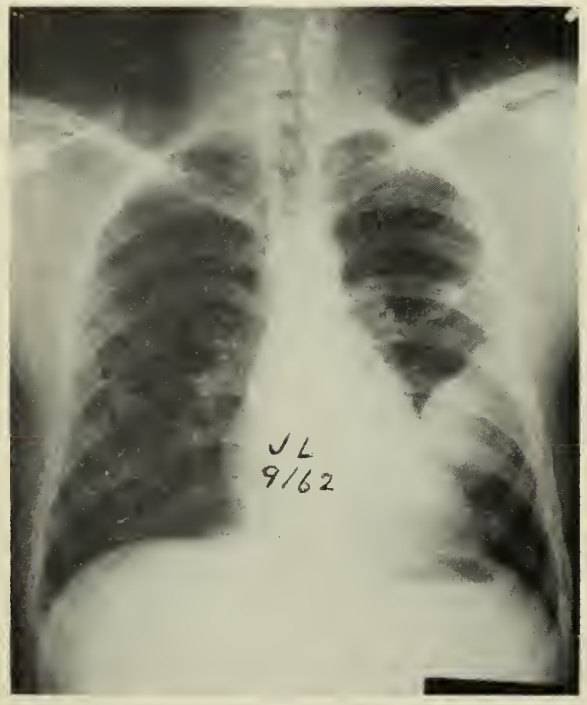


Fig. 4. Coin lesion right chest. Residual operative scarring on left



Fig. 5. Left pneumonectomy. Metastasis now in right upper lobe

tion of the lingula with a portion of the overlying pleura and rib. The tumor was friable and about 3 cm. in diameter.

Microscopically, (Figs. 2, 3) the lesion was identical to the previously removed lesion of the forearm.

The patient improved until September 1962, when he complained of some tightness and pleuritic pain in the left chest. X-ray (Fig. 4) demonstrated the previous operative area in the left chest and a new metastatic lesion in the right lung.

The lesion on the right side was easily removed by a wedge excision. Although the surgery was simple, the patient had an exceptionally stormy postoperative course. The patient presented a shock-like picture almost continuously for nearly ten days. It occurred to us that perhaps there might have been some humoral element excreted from the tumor which had caused this unexpected course. We could think of no technical error which had caused this bizarre clinical picture.

The patient gradually improved until May of 1963. A mass recurred in the left chest nearly occupying the lower third of the left pleural cavity. The patient would not accept a left pneumonectomy. He did accept Mustargen® in the usual dosage intravenously, 0.10 of 1 mg. per kilogram of body weight per day for three days. In addition, a cardiac catheter was inserted into the left pulmonary artery and 10 mg. of Mustargen® was given in 1 dose. The patient received 3,000 roentgen units of cobalt 60 to the tumor over a period of nineteen days. The tumor demonstrated a reduction in size of perhaps 50% by X-ray. By September 1963, the tumor again showed signs of recurrence and a total pneumonectomy was proposed and carried out. The patient had a very smooth postoperative course.

In January of 1964, the patient complained of cough and anorexia and was short of breath on slight exertion. X-ray showed a recurrence in the right upper lobe (Fig. 5). He had been receiving Cytoxan®, orally, in the meantime.

During the four years follow-up, it was interesting that at no time did the patient show any evidence of metastatic disease to either the lymph nodes, bones, or central nervous system. Several metastatic lesions appeared in the skin and subcutaneous tissues about six weeks before his death in May 1964. Autopsy was not permitted.

SUMMARY AND CONCLUSIONS

1. Hemangiopericytoma is an unusual and interesting vascular tumor of unpredictable behavior.
2. A case has been presented, which arose from the right forearm, and metastasized to the lungs and skin.
3. Treatment consisted of conservative removal of the primary lesion, followed later by radical surgery, cobalt therapy, and chemotherapy for the pulmonary metastasis.
4. Earlier radical surgery of the primary might have been more effective if the patient had allowed it.

Dr. Alan Raftery, pathologist, and Dr. Huseyin Sarikaya, M.D., Mercy Hospital, Canton, Ohio.

This diagnosis was concurred in by Prof. McKay of the Laboratory of Surgery of Columbia University College of Physicians and Surgeons, who kindly reviewed the microscopic slides.

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Owen H. Wangensteen

J. ARTHUR MYERS, M.D.

Minneapolis

"It is not the man of formal medical pedagogy that attracts students; contrariwise, scientists are the magnets of these schools." Billroth, 1876

PREFACE

HAD YOU TRAVELED up the Norwegian Sogne Fjord near Øvre Ardal, in 1880, you may have met a young man on holiday from the Bergen Military School. He may have delighted you by pointing out the farm where he was born in the year 1857,* the high mountains, the waterfalls, the trout streams, but then would doubtlessly come the request: tell me of the United States. This interest brought the venturesome 24-year-old Ove Wangensteen to Modena, Wis., in 1881.†

In 1886 Ove, now Owen by adoption of the American counterpart, is found established in a Lake Park mercantile business. The versatile Owen kept one eye on the store, another on the operation of a farm a short distance from Lake Park, while a third interest occupied a considerable portion of his time. The Thorvald Hansen family, immigrants from Norway, homesteaded near Lake Park and Owen's interest in their daughter, Hannah Mathilda, grew into a happy marriage Feb. 9, 1893.‡

OWEN H. WANGENSTEEN: YOUTH

Early (Norwegian) settlers led a simple and rugged life.§ There was always an abundance of work to sap the energies of the most active child; between school work and farm duties there was little time for the pleasures of hunting and fishing.

Owen, Jr., seemed to have the chore of milking the cows while more elusive brothers¶ did the hunting. He has commented of this experience saying, "We shared the profits. They learned to hunt and I learned to work. I sometimes think I got the best of the deal."[1]

Sadness entered young Owen's life with the death of his mother when he was but seven years old.**†† The family continued its close family circle guided by a stern yet loving Norwegian discipline which carried it firmly through rough times.

Unknowingly, but more deeply imbedded with each year, a trait of character developed in young Owen which today in sophistication he names "consecutiveness." As a youngster growing up it was an overwhelming pride not only in the completion of a task but completion with such perfection that it taxed his every ability. Neither time nor effort was to be counted to achieve a goal.

Owen had a great love and sympathy for farm animals. He now tells that "to have a hand in helping plants and animals grow filled me with a sense of the joy of living, of being useful, and of being a part of an important scene. It is still a pleasant dream."[2] "Frequently as quite a small boy returning from a long day's work in the field, almost too exhausted to remove the harness from the horses, I found it much easier to support my own fatigue as I thought of how tired

This is an introduction to a group of articles planned for publication in future issues of THE JOURNAL-LANCET under the title Owen H. Wangensteen Series.

*Ove (Owen) Wangensteen, Aug. 11, 1857-Dec. 5, 1937; son of Tollef Ove Aabölle Wangensteen and Kristi Hermundsdatter Kinsterdal

†Attended LaCrosse, Wis., Business College and St. Olaf College, Northfield, Minn., to perfect his English and learn bookkeeping

‡Thorwald Hansen, Hakhedal, Norway; Kristine Halvorsdatter Rasch, Hallingdal, Norway, met on ship bringing them to U.S. in 1867, married 1872.

§Biographical data obtained from relatives

¶Charles Thomas, May 8, 1897, attorney, Chisholm, Minn. Owen Harding, Sept. 21, 1898, surgeon, U. of Minn. Sister: (Christine) Marian, Dec. 2, 1899, Mrs. Chris Holm, Hibbing, Minn. Theodore Hanna, Aug. 26, 1902, attorney, Minneapolis.

**Died Dec. 20, 1905. Father married Miss Emma Kins, Modena, Wis., Aug. 31, 1910. Daughter: Alvina Kathryn, Dec. 9, 1912, Mrs. Jens Fossum, Fargo, N.D.

††"My mother had died when I was seven. My teacher, Miss Hansen probably knew how much a young boy needed his mother. As I look back now, Lizzie Hansen's words of encouragement no doubt reflected the wise discernment of a great teacher endowed with fine maternal instincts which she lavished warmly and selflessly upon succeeding sixth grade pupils over many decades. After more than 50 years thoughts of Lizzie Hansen bring back many pleasant memories, a tightness in the throat and moisture in the eye." *Jour. of Medical Education,* Vol. 37:701, July 1962. "Our Teachers."

those horses must also be."[3] This very idea, now in a far different setting is echoed by surgery residents as Dr. Wangensteen outlines a seemingly impossible task for them to do—and their comment is always, "he'll work at it twice as hard as we."

The story of how a young man's ambition to be a farmer was changed by the discovery that his hand was more facile as a delivery instrument for the birth of pigs is a humorous tale. Its significance lies in the recognition by a father who was close to his son that here was a boy who had ability which far outreached the home farm; possibly it could be medicine. Owen went to the university but he still thought of farming as a career.

Having won half his point, the father did not argue and Owen matriculated in the fall of 1915. A second well-known story of a summer vacation and having to haul manure on a succession of hot summer days causes a smile, but even so lowly an occupation could not have swayed a man whose sole ambition was farming. After much adamant persuasion, father and son reached a compromise. Owen still thought of farming or some variant thereof, but his father won out. Owen would have a go at medicine.

There is no doubt that the last summer's vacation on the farm nudged an already weakening decision in favor of a fascinating new interest which had developed through the auditing of some surgical lectures. In 1918 Owen entered Medical School, received his Bachelor of Arts Degree in 1919, and the Bachelor of Medicine Degree in 1921. The short period was due to the accelerated program in medicine during war and early postwar years. He did his Internship at University Hospital, and received the Degree of Doctor of Medicine in 1922.*

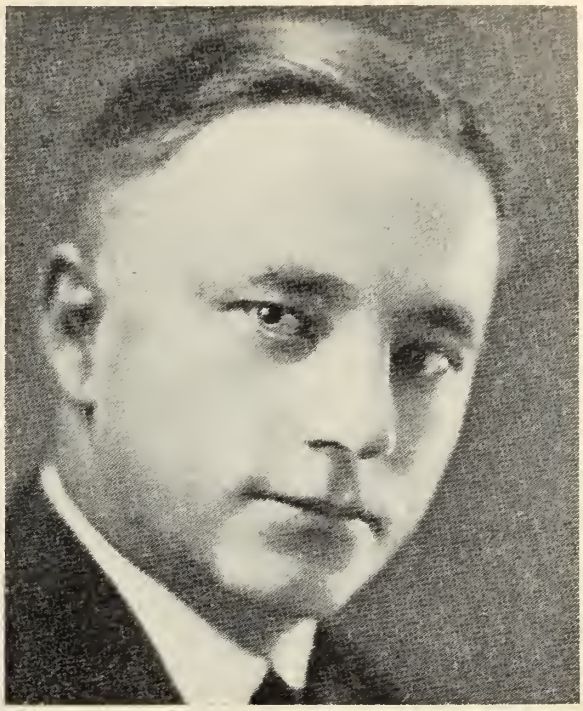
In spite of the fact that scholastically he held the highest standing in his class,† his future course still vacillated; no fellowship was available in surgery so he accepted a fellowship in the Department of Internal Medicine as a prelude to surgery. This fellowship carried with it an enormous drawback, and it was not the $600 stipend but the fact that each recipient must work out a research problem in the laboratory. Forced into research, submitting reluctantly, he discovered it an absorbing interest, an interest

*There had been no physician in recent generations in either family. However, Ove occasionally called attention to Ove Vangensteen as translator of Leonardo da Vinci's medical works and to Gerhard Armauer Hansen who had discovered the bacillus of leprosy.

†Elected to Alpha Omega Alpha, Sigma Xi

‡Thesis "The Undescended Testis, An Experimental and Clinical Study"

§Past president, American Medical Association



Dr. Wangensteen while a Fellow in Surgery at the Mayo Clinic

which foreshadowed the years to come. He spent the year 1924 at the Mayo Clinic and returned in January 1925 to complete the work toward a degree of Doctor of Philosophy in Surgery.‡

Dr. Leonard Larson,§ a classmate, has said, "when Owen took his Ph.D. examination, the examiners finally gave up. They couldn't ask him anything he couldn't answer."

When Dr. Wangensteen received the degree of Doctor of Philosophy he was offered a $15,000 a year job, but he decided to stay at the university at a salary of $3,600.[4]

SURGERY DEPARTMENT APPOINTMENT

When Dr. Strachauer resigned as Chairman of the Department of Surgery, University Hospitals, in 1925, Dean Lyon of the Medical School appointed a Search Committee whose duty it was to find and recommend Dr. Strachauer's successor. Candidates for the position included the late Drs. Francis Newton of Harvard School of Medicine and Mont Reid of the University of Cincinnati who had completed his training in surgery at Johns Hopkins School of Medicine under Professor Halsted.

Dr. Newton came to Minnesota to inspect the Department after which he said, "There is nothing worthwhile here nor will there ever be."[5] Dr. Reid agreed. Concerning this appraisal in later years, Dr. Wangensteen said: "It is true

that we didn't have much in the way of impressive buildings or funds in those days, but there was a certain atmosphere that our visitors overlooked."[6] He referred to a feeling for academic research nurtured by a few farsighted men at the University. Following suggestions of no interest in the headship by two surgeons who had examined the Department's future Dean Lyon, who was a keen observer with an uncanny ability to evaluate the qualities of young teachers, and members of the Administrative Committee of the Medical School had been so favorably impressed with Dr. Wangensteen's work from the time he entered the University in 1915 that, on Dec. 13, 1926, the Administrative Committee consisting of Dean Lyon, C. M. Jackson, J. C. Litzenberg, and F. W. Schlutz concluded that Dr. Wangensteen be selected to become the ultimate head of the Department of Surgery on a full-time basis. For a man with the rank of Instructor this was an unprecedented appointment. Dr. Wangensteen was never apprized of this situation and read it many years later in the minutes with a good deal of surprise. Dean Lyon urged that the young protégé have further preparation, a period of study in Europe, before assuming the Chairmanship. Dr. Strachauer delayed his plans for retirement five years, until 1930, continuing as Chairman until Dr. Wangensteen was ready to take over the Department. A financial provision of $2,400 was made and Dr. Wangensteen was elevated to the rank of Assistant Professor. During 1927-1928 he served as voluntary assistant in the Physiological Institute of Professor Leon Asher in Berne, Switzerland, while attending the surgical clinics of Professor Fritz de Quervain, Theodor Kocher's successor. When he returned, promotions followed quickly: an Associate Professorship in 1928, Surgery Department Chairman in 1930, and full Professorship in 1931.*

Concerning those who had served the School of Medicine and its Department of Surgery prior to 1930, Dr. Wangensteen has said: "One cannot read of the multiple facets concerned with the genesis of the Medical School without recognizing the sense of dedication, and the great interest of the founders, in creating a representative school with a strong staff."[2]

When Dr. Wangensteen became Chairman of the Department, Dr. Strachauer remained as Professor on part time. In 1930 there were only three other full-time clinical teachers in the Department of Surgery—Dr. O. J. Campbell for approximately one year, Dr. William T. Peyton, who became Director of the Division of Neurosurgery, and Dr. C. D. Creevy, then a Fellow and in 1932, a staff member, and later Director of the Division of Urology. There was only 1 fellow in 1929 when Dr. Wangensteen advised adding another. In 1931 there were only 6 fellows* in Surgery. Dr. Charles H. Mayo was a full-time Professor of General Surgery and came from the Mayo Clinic to give an occasional lecture. Additional teachers were recruits from private practice who graciously gave of their time to the Department. There were 7 Clinical Associate Professors and 10 Assistant Professors. Therefore, the new Chief of Surgery was surrounded by a splendid group of surgeons who were devoting a goodly portion of their time to lectures, operative procedures, and the outpatient clinics. Their help was incalculable to the growing Department.[5]

While preparing for the Headship, Dr. Wangensteen gave a great deal of thought to the organization of the Department. He formulated certain broad principles to which he continued to adhere. He says, "My thought has been that the power of appointment is the only authority one needs to guide the destinies of any venture. My judgments of whom to elevate to departmental responsibility have been influenced and decided largely by considerations of the available person's motivations, competence, interest in research and teaching, and likely contribution to the overall enterprise."[5] "Good men, I feel strongly, can be trusted and must be left alone to work out their salvation. A far more useful function is served, I am very certain, if the director of an enterprise abstains from trying to adjudicate or 'quarterback' every decision . . . Any teacher who fails to recognize that in some respect a large number of the trainees in his organization acquire skills or knowledge which make them his master is not alert to the potential power of the educative process. The professor's most important role . . . is to create or help synthesize an atmosphere in which learning becomes an absorbing, engaging and fascinating adventure and experience. In such a receptive environment industry and scholarship thrive bringing out latent talents and the finest quality in men. This is the first lesson of the Educator. The men and the proliferation of ideas emerging

*Professors of Surgery date back to the organization of the School of Medicine in 1888, when Dr. Perry Millard was appointed Dean and also Professor of Surgery; died 1897. Followed by Dr. Charles A. Wheaton 1897-1901, Dr. James A. Dunn 1901-1904, Dr. James E. Moore 1904-1909 and Chairman, Surgery Department 1909-1918. Dr. Arthur C. Strachauer, Acting Head 1918-1920; Head 1920-30

*Rolla Stewart, Charles Mead, Melville Manson, Carl Rice, Logan Leven and Herbert Carlson

and generated in such a milieu will prove to be the professor's greatest discovery."[5]

Dr. Wangensteen would be the first to deny that he has always, and everyone else has always, been in agreement with him, or he with them. In his paper on the Career of a Surgical Scholar Dr. Visscher[3] says, "Owen Wangensteen has not been a noncontroversial figure; but when he has engaged in a battle he has done it on the basis of conviction as to a principle, not for petty, selfish reasons." Dr. Wangensteen has said, "In facing up to criticism, I believe there are two correctives: patience and selfless dedication. These two can overcome any reasonable criticism which is not obstructive. To the occasional member of my Department venting his spleen or invective upon its activities, I always write or say, 'knowing you to be a responsible member of this Department and how much you value your appointment and your association with the University, I know you would not offer any other than constructive criticism.' It has been astonishing how this simple statement has sufficed to silence critics who had no constructive criticism to make."[5]

From an annual departmental budget of $30,000, which was far too small for Dr. Wangensteen's contemplated organization, the budget has grown to a figure well over 2 million dollars (which includes much Federal money) and is still considered too small. In order to increase his budget permitting the acquisition of more Fellows, he conceived the idea that the part-time clinical teachers turn their honoraria over to the Department. Dean Lyon advised taking it more slowly and going forward cautiously with the program. All part-time clinical teachers had private practices and Dr. Wangensteen felt they did not need their nominal salaries as much as did the Department of Surgery. This increased the departmental budget by $12,000. Such an unheard-of procedure precipitated a call from the Dean of Medical Science. However, Dr. Wangensteen's friends, President Lotus D. Coffman, Dean Elias P. Lyon, and Dr. William J. Mayo lent their staunch support, thus making his position secure and reaffirming Dean Lyon's and Dr. Strachauer's confidence in him.

"Sometimes a young man needs to have friends rise to his defense, an occurrence which I found very reassuring and helpful during my early years in this Department, when a little sniping from without was condoned, and encouraged from within."[5]

Dr. Wangensteen has always persevered to accomplish whatever he deemed best for his Department and the School. The occasional loss



Dr. Wangensteen, Professor and Chairman, Department of Surgery, University of Minnesota Hospitals

of a skirmish never caused him to give up. "None of us can find much consolation in defeat, yet time not infrequently suggests there may have been gain in the loss. In life's struggle, loss of battles is common experience. It is, however, no reason for abandoning an important cause."[5]

TRAINING OF SURGEONS

From his long experience in the training of surgeons, Dr. Wangensteen has made observations which could result in marked improvement in both undergraduate and graduate medical education systems. In 1962 he referred to the long time involved in the training of physicians. He said, "We could knock off a year in the elementary school period and another for the gifted student during the high school period. I would also abolish the internship. The practical training of the clerkship period is such that a person is well enough oriented to join the training ranks of his chosen specialty, if only he had made up his mind what to do. For the person wanting to go into general practice two years of hospital training should suffice. But for the specialist, whether internist or surgeon he should be prepared to enter the fellowship or residency training of his choice, without the intervening internship. The internship, after all, in my opinion, is only a form of cheap slave labor. If it were not that our medical military authorities exempt interns from military service until completion of

the internship, I have the feeling the internship would have disappeared a decade ago. I believe it is on the way out."[7] He considered the rotating internship, he himself took at the University of Minnesota, as a waste of time. In his Passano award address he commented: "My only parting reminder to teachers is that young men are so eager and keen to learn, so ardent in their desire to make their own contribution to the patrimony of knowledge, that all a teacher need do is provide them with the opportunity, and not stand in their way. That essentially has been my small contribution to the Minnesota enterprise. But was not this the injunction of the Great Master to all stewards?"[3]

The graduate training program must be fitted to the man. For General Surgery the minimum is five years. Trainees, who decide to stay in the academic fold, usually take six or seven years of which one year is spent in a surgical research laboratory and another in one of the preclinical disciplines as Biochemistry, Microbiology, Pathology, or Physiology. In an editorial in 1961, Dr. Wangensteen said, "For centuries the proper preparation for the surgeon has been the study of Anatomy; for the internist, Pathology. Today, knowledge of both is still of great importance to surgeons. However, it is to the disciplines of Biochemistry, Immunology, Microbiology, and Physiology to which we currently look with great expectations for developments that will advance the science and the art of Surgery."[8]

In 1935 when Dr. Maurice Visscher, then a young physiologist, became Head of the Department of Physiology, Dr. Wangensteen found him not only willing but eager to participate in the teaching of Physiology to Medical Fellows in Surgery. Thus, Dr. Wangensteen began to send some of his fellows to Dr. Visscher for a year of special training. This combined program has grown to the point where many Surgical Fellows added a minor in Physiology, or a combined major, gaining a Doctor of Philosophy degree in both Surgery and Physiology. This same type of program has also materialized with the Departments of Biochemistry, Pathology, and Microbiology. Dr. Visscher points out that the Surgical Fellows have contributed greatly to his Department "because they are skilled in operative techniques, they have made possible physiological experiments we might not otherwise have undertaken. This joint Surgery-Physiology program has been a two-way exchange."[6] It has been noted with pride that during experiments in Physiology involving the induction of heart failure by arteriovenous anastomosis, Dr. C. Walton Lillehei, then a Fellow, became interested in cardiac surgery.

Dr. Wangensteen has so impressed upon his Staff the benefits of working in the laboratory that he has kindled such enthusiasm among his fellows that he has " . . . trouble keeping them out of the laboratory. Every man knows that if he comes up with something new there his future is assured."[6] "The young surgeon, who during his pursuit of a research problem, has learned to operate with consecutive safety upon the dog is a surgeon to whom operations upon man can be entrusted, after acquisition of an adequate experience on the wards. Society as well as Boards of Surgery will come to recognize the merit of this position."[8]

When he became Chairman of the Department of Surgery, the Mayo Foundation for Medical Research, which had been in operation for many years and where he had enjoyed some of his training, was appreciated by Dr. Wangensteen. His admiration for the Mayo Foundation has been abiding. In 1963 he wrote, "Establishment of the Mayo Foundation within the Graduate School of the University of Minnesota (1917) has come to have great meaning for graduate training, not alone on this campus but also at the Mayo Clinic in Rochester. For more than sixty years the Mayo Clinic has enjoyed a dominant influence in medicine the world over. Surgeons especially, and their patients too, have been greatly influenced in their selection of the therapeutic procedure by current practice at the Mayo Clinic. From one end of the country to the other, products of the Mayo School of training today reflect the Mayo attitudes and practices . . . The genius of the Mayo brothers was reflected in the creation and integration of a great teaching and training enterprise as a corollary of an extraordinary clinical service function, cushioned by an imposing and extensive research program.[5]

"Criticism against the Department of Surgery for not pursuing a more systematic plan of teaching periodically has been leveled by both students and faculty members. Perhaps our methods are somewhat unorthodox, yet, in the National Boards, the performance of our undergraduate medical students as a group in the examinations in surgery regularly has been amongst the best in our school and, more often, the highest turned in by students in any of the clinical disciplines.[5]

"My own feeling through the years has been that there is no special virtue in trying to 'cover the waterfront' by a systematic plan. The breadth

of most clinical disciplines today is such that no one can teach or absorb it all. Thirty-five years ago it was possible for an earnest graduate student to embrace the entire current literature of any clinical field by spending three or four hours in the library once a week. Today, if an equally motivated graduate student in Surgery were to read every waking moment, it would be manifestly impossible for him to encompass the literature. As in earlier times, much of the moving scene taught today will quickly become obsolescent and should not absorb the students' attention to the exclusion of matters of great historical importance which have survived the wreck and rust of time. Much in any discipline can be written on a single page of history. Separation of the wheat from the chaff can only be accomplished by familiarizing the student with the origins of contemporary thought. The orientation afforded by a judicious appraisal of present knowledge against the background of time must not be underrated. What greater stimulus to the imagination is there than an acquaintance with the past, which is the history of men and ideas? It is very important, I believe, to expose the student to men teaching from the fullness of their subject, declaiming with enthusiasm upon the themes they know the best. The opportunity to hear Wallace Cole, Logan Leven, Richard Varco, Walton Lillehei, Donald Creevy, Lyle French, John Moe, Claude Hitchcock, William Kelly, Yoshio Sako, Fletcher Miller, Joe Aust, John Perry, Albert Sullivan, Richard Lillehei, Ed Humphrey, and many others in a few lectures at their best, discussing experiences in those areas in which they are acknowledged leaders, must be a refreshing and stimulating experience for the student, who, whether listening to them or to Doctors Visscher, Armstrong, Barnum, Lazarow, Watson, Spink, Good, or other great teachers within the school will say to himself: 'That is the kind of person I would like to be. . . .' When the stimulating contagion of a consuming interest overtakes the student, boredom, floundering, and inertia are supplanted by enthusiasm, growth, maturity, and vision. Students are to be enticed, not driven, into the Elysian fields of scholarship, where the pursuit of knowledge can become an absorbing, engaging, pleasant, and soul-satisfying occupation as well as pastime. The compensations of the teacher are great. To be privileged to make indelible imprints upon alert, impressionable, and responsive minds, to enlarge the vision, or recreate the thinking of others—these are sources of abiding satisfaction. . . ."[5] "Observation, experiment and experience—these are the great teachers of us all."[9]

The Heads of other Surgery Departments visit at Minnesota and leave feeling that they have, for a brief time, come to know a great teacher. "It is always instructive and a great privilege to be able to visit your Department, and, of course, this was no exception. As with so many others, I look up to the wonderful path that you have trodden as the one I would so much like to follow."*

As a first year Fellow in Surgery, Dr. Wangensteen earned $600 per year, an income so meager that it left an indelible impression. He later spoke out, that "the time is by when the young House Officer is content to work for glory alone. He merits and deserves a living wage . . . If a student having completed his undergraduate period of medical school training embarks upon a career in a specialty, he cannot afford to prejudice the future by accepting loans."[7,10] "In 1960 a beginning salary of $3,600 may be a bit out of line with what is the current going wage for trainees, but it is in effect a low estimate of the value of such a person to the hospital and the department he serves. From there on to the completion of his training, he should, I feel, be given annual increases of $600 to $900. For the trainee anxious to complete a Ph.D. program with his eye on the academic ladder, advancement in the clinic surgically is always a bit slower, but it is this individual who especially merits more rapid increases in pay. It is my belief that if the pay schedule for our surgical fellows or trainees could be stepped up, all the worry and concern over completion of the training program in a 4-year period would disappear."[11]

During Dr. Wangensteen's Chairmanship, he has successfully made financial arrangements to provide support for both fellowships and research projects. Each application has been carefully reworked and reworked to fully justify the request for substantial funds to carry the project. Besides evaluation of his own projects, he has served on the committees of granting agencies and helped evaluate the projects, under the auspices of other universities.

Since 1937 Dr. Wangensteen has not sent statements for his services to his few affluent patients. When such persons inquire for their bills his reply has been, "You can give something for research if you like."[4] Since that time large

*Dr. Ben Eiseman, Prof. and Chm. Surg. Dept., U. of Ky. Jan. 6, 1966. Personal correspondence. Letter from Dept. of Surgery files to which the writer had access.

numbers of persons have contributed to the field of research in which they have a personal interest. Personal gain from patients has been small. For patients who have paid Dr. Wangensteen, he has always charged only a very modest fee. He has never charged a patient more than $400 and that patient had several procedures during one hospital admission. His usual fee has ranged from $150 to $250. From this modest emolument he further saw fit to establish, through cooperation with the hospital, a fund whereby the patient was charged a minimal bill and these payments went into a research fund, the Baker Fund. Several of Dr. Wangensteen's colleagues joined him in accumulating research funds for the laboratories in this way.

The Surgical Fellowship Training Program quickly became so superior under Dr. Wangensteen's direction that the number of applicants rapidly increased. So fast was the increase that his departmental budget was most inadequate. In 1960 he wrote, "Together with a few of my associates, I am continually on the prowl for likely sources of support, a responsibility which incites insomnia and creates the necessity for giving much thought during working hours to this modern-day problem of professors."[11] His phenomenal success in this enterprise is appreciated by those who know that of the 70 Fellows in General Surgery during 1966-1967 only seven are supported by State funds in the Departmental Budget. The remaining 63 are supported by United States Public Health Service Fellowships, Foundations, and Friends of the Department. In 1955 he expressed his appreciation of Dean Diehl's help by saying that under his aegis the Surgery Departmental Budget had increased 700%.[2] Since 1958 staunch support has continued under the approving eye of Dean Robert Howard.

STIMULATION OF RESEARCH

One resident physician speaking of going on rounds with Dr. Wangensteen said: "If you follow him with a pencil you can write down 20 original ideas each day."[4]

"To an inquiring mind, literally hundreds of subjects of daily concern suggest the need for investigation. Thinking, asking, listening, reasoning, reading, questioning, constructing premises, challenging them as well as the conclusions—these are the laborious and time-consuming techniques of the investigator. Maintenance of a childlike wonder is the best preparation of the mind for the investigator. In retrospect it is often astounding to see how simple the solution was. One finds oneself asking: why was this not thought of before? It is often a very proper question. We all have mental blind spots. Presently a fact or bit of information to which little or no significance has been attached, begins for the first time to have important meaning."[3]

In his Passano award address Dr. Wangensteen said: "My work essentially has been that of plumber of the alimentary canal. I have worked at both ends, but largely in between. My friends and colleagues tonight already have placed my work in a more favorable light than it deserves . . . Intestinal obstruction and its management, the nature of the peptic ulcer diathesis in its many manifestations, the origin of appendicitis and lithiasis of the biliary tract and surgical attacks upon the problems of alimentary tract cancer—these are essentially the items that have occupied my attention these many years. An occasional foray has been made into other sectors, but to these items I have invariably returned."[3]

"It interested me that when you put a catheter in the bowel lumen the obstruction sort of righted itself. I wondered what would happen if you put a catheter down the nose to remove gas and fluid from the stomach and intestines."[4] Concerning this one procedure, Dr. Visscher said in 1944,[12] that it had saved 100,000 human lives. From this gastric suction technique and equipment and for its administration alone Wangensteen became a household name in hospitals around the world.

He revised existing techniques of radical resection of the stomach and colon for chronic ulcer and malignancy, and in 1948 he initiated the "second look" procedure, that of exploring the abdomen at intervals of approximately six months until no trace of malignancy was found. In some cases this has been done as many as half a dozen times. By making subsequent reentries of inspection he has been able to salvage additional lives of persons with cancer of the stomach and 15% of patients with cancer of the colon who otherwise may have died from malignancy.[6]

"Many times I have been asked how the idea of using local gastric cooling for the control of massive gastric hemorrhage in patients with peptic ulcer came about. I have answered by saying the thought came from reading the papers of Langley of Edinburgh on factors influencing peptic digestion. Much to my surprise on rereading Langley's papers, I failed to find any suggestion therein concerning the influence of temperature on the digestive process. . . . For long years, the relationship of temperature to

enzyme activity had been known to biochemists as well as physicians. The only strange thing is that I had not considered the possibility 25 years ago, when first beginning to tinker with the stomach.

"Fortunately, we had available a critical method for direct testing in the cat's esophagus, which is very susceptible to injury of acid peptic juice. In fact, this technique has been used in our clinic for more than a decade to assess the potential digestive power of gastric juice. While sitting in the library with the Langley papers in my hand, I telephoned my associate, Dr. Harlan Root in the Laboratory, proposing the experiment to test the validity of the suggestion. The experiment was run simultaneously on cats under conditions of normal temperature and of hypothermia. In a few hours we knew the results. Perforation or evidences of severe esophagitis occurred in the cat's esophagus after 2 hours attending its perfusion with warm gastric juice obtained from a patient with duodenal ulcer. In the cooled cat, perfusion of gastric juice when cold, provoked little or no evidence of injury. In other words, reading of Langley's papers, which had not mentioned the depressive effect of lowered temperature upon enzyme activity, did suggest the idea of another mode of attack. This experience must be common to all experimenters. It points up the need for reading in the interphases of disciplines relating to problems under investigation."[3]

Gastric cooling has become an accepted procedure. There is no question of its role in stopping hemorrhage. The potential of gastric freezing in controlling the secretion of acid gastric juice and thereby controlling peptic ulcer diathesis remains in the proving stage.

AMERICAN COLLEGE OF SURGEONS: SURGICAL FORUM

In 1940 during the meeting of the American College of Surgeons, Dr. Wangensteen emphasized the need for a surgical forum, "one of the most urgent and obvious needs among surgical organizations in this country has been a Surgical Forum to which young men can bring their contributions and secure an audience."[13] In 1942 as chairman of the Committee he said, "When the American College of Surgeons convenes for its Annual Clinical Congress the second Surgical Forum on Fundamental Surgical Problems will be held also. This portion of the program, instituted by the Board of Regents, afforded a large surgical audience the opportunity to become acquainted with the work of many productive clinical and experimental investigators through the medium of the concise presentation; at the same time, it accorded a number of young, well-trained surgeons their first opportunity of a hearing before a national surgical organization. It is important that young, productive workers in surgery be provided an opportunity early in their careers for active participation in surgical gatherings—before the ravages of time dampen their enthusiasm."[14] In 1960 he wrote, "The Surgical Forum of the American College of Surgeons has played an important role, I believe, in emphasizing to all surgeons the great importance of the experimental method. . . . Today no surgeon can afford to neglect reading the *Proceedings of the Forum*. It is truly the Arabian Nights reader for surgeons. Moreover, a generation of surgeons nurtured on the 'Surgical Forum' and its *Proceedings* will enable American surgery to rise to a higher level."[11]

THE CANCER DETECTION CENTER

In 1948 the Cancer Detection Center began as the outgrowth of Dr. Wangensteen's and his colleagues' theory that the earlier any evidence of cancer is found, the ratio of patient survival increases. Since cancer becomes more prevalent in older people, the age of 40 or older was set as the minimal age for the study. Only asymptomatic adults were taken. A complete physical examination is given, any suspicious findings are reported to the person's own physician for diagnosis; the Center does not treat any patient. Participants return once a year. The Center averages approximately 5,000 examinations yearly.* During the initial examination, one possible cancer is found in 67 men, one in 88 women. During repeat examinations, over a five-year period, this ratio drops for men to one in 121 and for women one in 153.

With the interest created by the Cancer Detection Center, clinics have been requested and operated in several parts of the state. In these clinics, unsuspected cancers were found in 1.5 patients per 1,000 examinations.*

DIEHL HALL BIOMEDICAL LIBRARY

Since the Library of the School of Medicine was established in 1888, probably no member of the faculty has used it more than Dr. Wangensteen. When Dean Diehl announced in 1954 that a new medical library building within the Medical School Complex was among the list of new buildings he proposed to procure, Dr. Wangensteen was at the forefront to promote this build-

*Personal communication, Dr. Victor Gilbertson, Director of the Cancer Detection Center.

ing project.* That the Medical School have a library par excellence became his consuming preoccupation. In 1961 he mailed letters to his friends and supporters which contained one key paragraph: "A Library and a great collection of books stimulate the love of learning in an institution, which influence, in the final analysis, characterizes the atmosphere of a true University. The Library, I am very certain, will lend a great impetus to research and scholarship and to the cultivation, nurture and quickening of the entire learning process amongst undergraduate as well as graduate students. For me it has been a revelation to have the opportunity of observing how students grow and mature in an atmosphere friendly to learning, surpassing the accomplishments of their teachers. For me, it has been a thrilling and rewarding experience to enjoy the wonderful privilege of studying in the presence of great books, whose pages preserve the permanent legacy of great ideas. Many a medical student and graduate student in the many disciplines represented in the Library, I believe, will have their sights lifted and the nature of their dedication increased by the use of this Library. In the year and a half during which a portion of the Library building now under construction has been in operation, more than 750,000† visits of students and faculty have been recorded by the turnstiles. I have the feeling that frequent use of the Library will come to have a telling effect upon our students and our School. The atmosphere of a good Library works silently upon the mind, uplifting the spirit and enlarging the student's vision. Sometimes I like to believe it is like the stirring urge of the Divine at work within the student. Can you help us?"

On December 27, 1961, with the endorsement of Dean Howard, Dr. Wangensteen wrote all members of the Medical School Faculty from the Instructor level up to enlist their help and contributions to the library venture.

The result of this personal and self-revealing plea did bring in funds which were a very substantial help.‡ He was not alone in this endeavor and one of his warmest friends, Dr. Donald Cowling, spent many hours with him going over figures and contacting friends to see from where the next helpful impetus might come.

Dr. Wangensteen envisioned that there must not only be the medical handbook and current journal of every description, but the medical student must also be aware of his medical heritage. Through one benefactor* this section of medical reference was born. Other friends rallied and nurtured this new enterprise to the extent that the fifth floor of Diehl Hall now houses an enviable collection of rare books.

Dr. Wangensteen suggested that one area be set aside for ready access to the classics of all ages in all fields. The really well-rounded person must have a love of reading in all areas. The following quotation from Anthony Trollope was hung by Dr. Wangensteen in the Library as well as in his own office area:

THE PUREST AND THE MOST PERFECT PLEASURE

In reference to this habit of reading, I make bold to tell you (that) it is your pass to the greatest, the purest, and the most perfect pleasure which God has prepared for his creatures. Other pleasures may be more ecstatic, when a man looks into a girl's eye for love, and finds it there, nothing may afford him greater joy for the moment; when a father sees his son return after a long absence, it may be a great pleasure for the moment; but the habit of reading is the only enjoyment I know, in which there is no alloy. It lasts when all other pleasures fade; it will be there to support you when all other recreations are gone; it will be present . . . when the energies of your body have fallen away. . . . It will last to your deathbed and it will make your hours pleasant while you live. But, my friends, you cannot acquire the habit in old age; you cannot acquire that habit in middle age; you must do it now, when you are young. You must learn to read and like reading to make reading a pleasurable pursuit while you are young, or it cannot become so when you are old.

Now that the Library has its complement of books steadily growing, Dr. Wangensteen foresaw a Medical History Department. To this end through his encouragement, friends were providing endowment funds. A History of Medicine Professorship was recently endowed by one of Dr. Wangensteen's patients.† A search was under way for a strong person to fill this vital Chair whose interest and personality would be such

*It was then thought that matching monies from the Federal Government would be available. However, this was found impossible because most of the 80 medical schools in the country came forward with similar requests. Therefore, all such government requests were denied. In 1955 the State Legislature appropriated $400,000 and the same amount in 1957. Although ground was broken for the new library building in June 1958, building was curtailed from time to time for lack of funds through 1961.

†Turnstile library record August 1966 was in excess of 2,500,000

‡In his letter Dr. Wangensteen pointed out that generous help toward the Medical Library had recently come from Mr. O'Shaughnessy, who gave $225,000 and an anonymous donor who gave $300,000 toward the Medical Library.

*Mr. John Pillsbury Sr., $100,000

†The late Miss Grace Polk

that he will make the knowledge found between bindings a living and essential part of each student's training. Mr. Leonard G. Wilson of Yale has been appointed to this Professorship beginning July 1, 1967.

PUBLICATIONS

It may be said that Dr. Wangensteen is the author of the "Bible" on intestinal obstruction. In 1935 he was awarded the quinquennial Samuel D. Gross Prize by the Philadelphia Academy of Surgery on his essay entitled "The Therapeutic Problem in Bowel Obstructions" and inscribed with the motto "a good name is rather to be chosen than great riches" (Proverbs 22:1). One of the stipulations of the Award was that the essay appear in print. This essay constitutes Part I of a monograph published by Charles C Thomas in 1937 under the title "The Therapeutic Problem in Bowel Obstruction, A Physiological and Clinical Consideration." The dedication reads: "To my Father and the Memory of my Mother in filial affection and devotion."

This book of 360 pages was so popular that a second edition was demanded; the first printing was done in May 1942. Second, third, and fourth printings were issued in August 1945, February 1947, and October 1949, respectively. By demand a third edition was printed in 1955 under the title "Intestinal Obstructions: Physiological and Clinical Considerations with Emphasis on Therapy, Including a Description of Operative Procedures." This consists of 838 pages. The dedication reads as follows: "To the many surgical fellows in the Department of Surgery at the University of Minnesota whose contributions and labors during the past quarter Century have added knowledge and accomplishment to the understanding and management of the bowel obstruction problem . . . to these many good colleagues and friends, this monograph is dedicated in the spirit of gratitude, affection and admiration."

In 1951 Dr. Wangensteen wrote a monograph entitled "Cancer of the Esophagus and the Stomach" for the American Cancer Society. It was so popular that a second edition was published in 1956 which is still continually in demand.

When the present *Journal-Lancet* Editorial Board was being organized in 1929, Dr. Wangensteen was at the top of the list of those invited to membership. Throughout the years he has been one of the most active and effective supporters of the Journal, having procured numerous articles for publication, having published several of his own manuscripts, and having produced and secured manuscripts for entire special issues.

In 1937 Dr. Wangensteen was chosen from the surgeons of America as co-editor of a new journal named *Surgery*. This journal soon became standard in medical and hospital libraries.

Dr. Wangensteen has encouraged members of his Staff and Fellows to prepare and publish manuscripts containing valuable information. From the Department of Surgery more than 3,000 papers have been published since Dr. Wangensteen became Chairman in 1930. Independently, and in cooperation with his staff, he has amassed a bibliography of over 600 published papers.

JAMES E. MOORE SOCIETY

In 1961 Dr. Wangensteen was instrumental in organizing the James E. Moore Society, an Honor Society of our Medical School. He states that Dr. J. B. Aust has been largely responsible for the activities of the Society. At the inaugural meeting, held on November 21, 1961, Mr. Ronald Dietzman, a senior medical student and founding member of the Society, presented a manuscript later published under the title "James Edward Moore, Educator and Surgeon, His Life and Work."[15] Dr. Moore was the first full-time Professor of Surgery in the Medical School. The Society is limited to 25 Junior and Senior Medical students. Eleven new members are selected each year from the Sophomore Class on the basis of scholarship, research interest, or specialized talent. In 1964 the Senior members were presented at the Medical School Recognization Ceremonies, firmly establishing the James E. Moore Society as an honor society of our Medical School.

RECOGNITION OF CONTRIBUTION TO STUDENTS AND UNIVERSITY

On September 23, 1955, Dr. Wangensteen's former students joined with more than 700 of his friends and surgical colleagues to observe the 25th Anniversary of his appointment as Professor and Chairman of the Department of Surgery at the University of Minnesota.*

The plan for this recognition began in the spring of 1953 when former surgical residents founded the Owen H. Wangensteen Surgical Education Foundation constituted to "further objectives that will serve as suitable tributes to Dr. Wangensteen." The enthusiastic support resulted in a fund adequate to provide for travel

*Committee Chairman, Dr. Claude Hitchcock

and educational opportunities for Fellows in Surgery at the University.

At this celebration Dean H. S. Diehl said, "This occasion honors you at the height of your career and honors you for your distinguished achievements as a surgeon, as a scholar, as an investigator, as a teacher, as a leader, as a wise and stimulating counselor and as a friend." He spoke of Dr. Wangensteen's modesty in his requests for the Department of Surgery, how he earnestly and convincingly presented the needs of his Department, but in so doing he had never placed the interests of the Department of Surgery above the interests of the Medical School as a whole.

Asked to speak on "A Record of Distinction," Dean Diehl questioned "Just what yardstick one should use in measuring Dr. Wangensteen's achievements, . . . the most important contributions for relief of intestinal obstruction following abdominal surgery; the surgical treatment of gastric and duodenal ulcers; his pioneer work and continuing interest and encouragement in the field of heart surgery; his distinguished achievements in the field of cancer surgery, . . . and the many improvements which he has made in surgical techniques and procedures. . . . What I would emphasize, however, is the great and unique contribution which Dr. Wangensteen has made in firing the imaginations and in training a multitude of able young men. It is this ability to inspire and develop young men to which I would pay special tribute. It is an ability which great teachers possess and through which they gain a type of immortality."[2]

The same evening John M. Russell, Executive Director and Vice-President of the John and Mary R. Markle Foundation, New York City, said: "Owen, I have read what the books have said about you, I have heard what the scientists have said about you, I have heard what the surgeons think of you, and I have talked to dozens of your students. With all that has been said today and with all that will be said tomorrow, I come at last to one very simple thing, nothing you have done, no matter how important, exceeds your capacity to be a friend. So, on behalf of all your friends who are here, and those who couldn't get here, I would like to offer this toast: Hail to the Chief—a really great friend!"[2]

President Morrill added: "This occasion we know is the tribute of his professional colleagues to Dr. Wangensteen. But, its climate is that of the campus as well and of the University which his distinguished endeavors have dignified. And beyond the boundaries of the Medical School on the campus, the rest of us—his University Colleagues—cherish the honor of privileged association with him and of pride in the institutional integrity which his brilliant career illuminates."[2]

In response Dr. Wangensteen said: "Praise always falls gently and pleasantly on the eardrums . . . That you should look with so approving an eye upon the accomplishments of my Department pleases, but embarrasses me at the same time, for I am only too aware of how many an opportunity for the University and the Medical School slipped through my hands unrecognized, and how many more were not fully utilized, that not infrequently I occupied myself with thimble sized matters when momentous problems, begging for a solution, went unnoticed."[2]

Dr. Wangensteen has been the recipient of numerous awards including the National Academy of Science. However, space permits special reference to only one—The Passano Award. Dr. Leonard W. Larson, President, American Medical Association, in presenting the Passano Award on June 28, 1961, to Dr. Wangensteen, its seventeenth recipient, said: "Each new name added to this honor roll gives recognition and appreciation to another of those individuals who can, through his own ability, spirit and vision, pass on to others the knowledge and inspiration needed to help solve our present day problems, and hasten the promise of tomorrow. Such a man is Owen H. Wangensteen. We need his example as we need his influence to continue the advance of medical knowledge through the chain reaction of learning, and of medical practice through the strength of dedication."[3]

Dr. Maurice Visscher added: "A 'complete' scientific scholar must have at least 3 important attributes: He must be devoted to the search for knowledge; he must be concerned with the reproduction of his kind; and he must be a statesman in his relation to his institution and to society. A signally successful, complete scientific scholar must have been extraordinarily competent and extraordinarily diligent in all three areas. No working scientist I have ever known has done much unless he has kept himself as productively busy as the proverbial bee throughout his working life. Certainly, Owen Wangensteen has done so."[3]

In his classical response, Dr. Wangensteen commented: "At the outset let me say the Passano Award in this instance belongs as much to my school as to me, and to my colleagues whose labors in research have done much to bring recognition to the Medical School of the University of Minnesota . . . In life's daily

drama it is undoubtedly persons who cogitate upon problems, feel deeply about them, who keep them continually in mind, and who are willing to work for their solutions, who make the greatest impact upon their own times . . . Life presents very few bridges to help us over the morass of daily confusions, difficulties, and obstacles in our climb, each of us must erect his own 'Jacob's Ladder.' There is no escalator to success."

"Every generation honored by succeeding ages has erected its own monument, not of marble but in accomplishment. The only leadership is to excell, the only way a contest can be won is by outpointing the challenger in performance. If we as a people are to maintain a position of leadership we must outthink and outproduce our rivals in Science, Industry, Agriculture and in the Arts. We must set goals and strive with determination and dedication to meet them.

"To bring out the latent power in a student is perhaps the most important function and achievement of the teacher. Every man has talents never used to capacity . . . Loss of human talent and power is the great waste of our time and deserves respectful attention of educators and government.

"As the life of a parent continues in a child so too the life of a teacher goes on in his pupils. As a father thrills in having a son whose achievement dwarfs his own, so too, the teacher finds as great and keen satisfaction in the accomplishment of his scholar sons for whose training he was partly responsible as in any accomplishment of his own."[3] "The only real gift in life is to give a part of oneself."[4]

THE SURGERY DEPARTMENT TODAY

Dr. Wangensteen's accomplishments are so numerous as to require a large volume by a professional biographer to do them justice. His ability to organize is exemplified by the growth and quality of his staff. From the small staff, only three of whom were on full time in 1930, the number has increased so that in his final year as Chief of the Surgery Department the roster lists: 13 full-time professors, 1 Professor Emeritus, 12 Clinical Professors, 6 Clinical Professors Emeritus, 4 Associate Professors, 23 Clinical Associate Professors, 6 Clinical Associate Professors Emeritus, 13 Assistant Professors, 50 Clinical Assistant Professors, 1 Clinical Assistant Professor Emeritus, 9 Instructors, 72 Clinical Instructors, 1 Clinical Assistant, and 1 Research Associate—making a total of 167 Faculty members.

In addition (in August 1966) there are 180 surgical Fellows, of whom 93 are at the University Hospital, 15 at St. Paul–Ramsey Hospital, 2 at Gillette Hospital, and 24 at the Hennepin County General Hospital, 43 at the Veterans Administration Hospital, and 3 at Mount Sinai Hospital. In the Department of Surgery since Fellowships were established, 108 Fellows have earned the degree of Master of Science. In addition to those awarded advanced degrees, there has been an even larger number who took graduate training of shorter duration and did not remain long enough to earn degrees. An increasing number of Fellows from abroad came for shorter periods of training. The current year has representation from 21 foreign countries.

The quality of training received in Dr. Wangensteen's department is reflected by the positions the former trainees now hold. Thirty-five are full-time professors of surgery or heads of departments of surgery,* while 30 others have full-time academic positions of lower rank. Eleven surgical trainees have won Markle Scholarships. More than 100 former surgical Fellows are engaged in some kind of academic teaching responsibility.†

In 1965 Dr. Wangensteen said: "The future of Medicine and Surgery demands that while we strive to improve the science of our craft, we do not neglect the compassionate graces of sympathy, mercy and charity."[16]

Dr. Wangensteen's positions, memberships, honors and advisory councils are too numerous to describe in this manuscript; therefore, only the following listing is presented:

Appointments, University of Minnesota:

Chairman, Dept. of Surgery, University of Minnesota Hospitals, 1930

Professor of Surgery, University of Minnesota Hospitals, 1931

Distinguished Service Professor, 1960

Chairman, Medical School Committee on Medical History, 1961

Regents Professor, 1966.

Scholastic Societies:

Alpha Omega Alpha, Sigma Xi

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*Two trainees who became Heads of Surgery, namely, Clarence Dennis and Bernard Zimmerman, are sons of former teachers in the Department of Surgery. Dr. Wallace Ritchie, who became a Fellow in the Department of Surgery in July 1963, is the fourth generation of Ritchies identified with the School, tracing down from Parks Ritchie, who was Dean and Professor of Obstetrics, to Harry P. Ritchie, Clinical Professor of Neurosurgery.

†In the July 1964 issue of *Surgery*, Dr. E. H. Ellison wrote concerning current surgical department chairmen in schools of medicine in the United States, pointing out that the most numerically productive department in recent years has been the University of Minnesota which has eight of the current professors and chairmen of departments of surgery. Since then an additional one was added making a total of 9.

Military Service:

Student Army Training Corps, 1918

Consultant to Office of Research and Development of National Research Council, 1939-1945

Consultant to Surgeon General U.S. Army in Germany, in summer of 1959.

Honors:

Samuel D. Gross Prize, City of Philadelphia, Philadelphia Academy of Surgery, July 25, 1935

John Scott Medal and Award, City of Philadelphia, May 15, 1941

Honorary L.L.D., University of Buffalo, N.Y., 1946

Alvarenza Prize, the College of Physicians of Philadelphia, November 9, 1949

Honorary D.S.C., University of Chicago, 1956

Pittsburgh Surgical Society Award, May 28, 1958

Honorary Doctor of Science, St. Olaf College, Northfield, Minn., 1958

Medallion and Membership in the French National Academy of Medicine, 1959

Corresponding Member German Surgical Congress, 1960

Passano Award, June 1961

Honorary Doctor of Science, Temple University, 1961

Honorary Fellow Royal College of Surgeons of England, 1962

Honorary Member Hellenic Surgical Association, Athens, Greece, 1962

Docteur Honoris Causa, University of Paris (Sorbonne), 1962

Special Citation, American Cancer Society, November 12, 1962

Honorary Doctor of Science, Hamline University, St. Paul, Minn., 1963

Honorary Member Norwegian Academy of Science, March 1964

Honorary Fellow Royal College of Surgeons, Edinburgh, 1965

National Academy of Science, April 1966.

Boards and Advisory Councils:

Office of Scientific Research and Development, 1940-1945

Gastric Cancer Committee, United States Public Health Service (Chairman), 1948-1951

Co-Editor of *Surgery*, 1937 to present

Surgical Forum, American College of Surgeons, Chairman, 1941 to 1952

Committee on Growth, National Research Council, United State Public Health Service, 1947-1949

Board of Regents, American College of Surgeons, 1950 to November 1, 1956

Surgery Study Section, United States Public Health Service, 1949-1953 (Chairman, 1952 and 1953)

Board of Directors, American Cancer Society, 1952 to 1957

National Heart Council, U.S.P.H.S., 1953 to 1957

Research Facilities Council, U.S.P.H.S., 1958-1960

Advisory Committee to Heart Council, 1960-1963.

EPILOGUE

Just as the bird must fly from the nest, the child leave the protection of home, so the young surgeon must find his horizons to conquer. The love and devotion, the respect and admiration for and the dependability of their chief have often been expressed by those leaving the Department. One such expression is as follows:

"As my time of departure from your Department draws near it is with real regret that I shall no longer be working under your aegis. I can only say that I shall be forever grateful for your encouragement and wisdom in directing my surgical training and research. Each of us is influenced by our contact with great men and I realize ever more fully my great good fortune to have worked for you. I suppose that in the future many men will claim to have been one of Wangensteen's Residents to bask in the aura surrounding such a privilege, as has occurred for Halsted, Billroth, and Cushing, but I am proud that I shall be able to claim honestly such an honor."*

It has been said that when one door closes another opens. Though the door of the elder Wangensteen must perforce close, another Wangensteen, the younger, has entered the surgical arena.†

---

*H. D. Root, Assistant Professor, April 1, 1966, personal correspondence.

†Stephen Wangensteen, Assistant Professor, Surgical Department—University of Virginia, Charlottesville, Va.

The author wishes to thank Miss Dorothy Riley and Mrs. Leontine Hans for their most valuable assistance in the preparation for this manuscript.

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Book Reviews . . .

The Peripheral Nervous System

by J. P. SCHADE, *230 pp., ill., Elsevier Pub. Co., New York, 1966. $10*

In the preface the author states that the book was written to fulfill the need for a simple and comprehensive introduction to the study of the peripheral nervous system. If a lenient definition of the word comprehensive is allowed, the author does achieve this objective. The themes of the book are brevity and simplicity. The text consists of a series of short paragraphs. Each describes the course of one of the peripheral or cranial nerves and a method of clinically testing the strength of the innervated muscles. Some clinical correlations between anatomy and peripheral nerve injuries are made, but these are too abbreviated to be used for study purposes. The simple format allows for easy relocation of desired information.

The text is amply illustrated. Each nerve discussed is shown in its course to the appropriate skin areas or muscles. The muscles are represented as shadows superimposed upon an outline of the skeleton. This approach is useful for depicting a general idea of the anatomy. It does not convey the true spatial interrelationships of the muscles. There are three large foldout plates which summarize much of the illustrated material. These are contained in a pocket on the back cover where, unfortunately, they run the danger of being lost.

The practitioner or student will find the book to be a useful outline for orientation in the study of the anatomy of the peripheral nervous system. It is the type of reference which can be used quickly when a question arises in the physician's office while he is seeing patients. The illustrations can be used by the physician as visual aids during his explanations of disease processes to his patients. The book should not be expected to give the more intricate anatomical relationships which are necessary for making a detailed muscle examination or planning a surgical approach. At $10 the book is slightly overpriced.

WILLIAM R. KENNEDY, M.D.
Minneapolis

The Artful Practice of Medicine

edited by WILLIAM B. D. VAN AUKEN, M.D., *115 pp., Charles C Thomas, Springfield, Ill., 1966. $6.50*

This treatise represents a commendable effort on the part of the author to record the accumulated wisdom of his nearly fifty years in medicine. Practicing in an age when medicine was admittedly more art than science, he deplores the present trend toward purely scientific mechanical medicine to the exclusion of "artful practice."

One chapter is entitled "Medical Ethics and Stuff," which could have been the title of the book. The organization of the various chapters is somewhat haphazard, but eminently readable and replete with many personal anecdotes, humorous stories, cliches, and platitudes. Much of the advice contained in the book is specific rather than general and in some instances may contradict current medical practice: for example, Dr. Van Auken prefers to dispense drugs in most instances rather than write prescriptions.

Directed primarily to senior medical students, interns, and beginning practitioners, the book contains many pearls of wisdom from a long medical career and is well worth the approximate hour it takes to read.

EDWIN O. HIEB, M.D.
Jamestown

The Stomach (Including Related Areas in the Esophagus and Duodenum): The Thirteenth Hahnemann Symposium

edited by CHARLES M. THOMPSON, M.D., DONALD BERKOWITZ, M.D., EDWIN POLISH, M.D., *and* JOHN H. MOYER, M.D., *474 pp., ill., Grune & Stratton, New York, 1967. $23.50*

This volume, with its up-to-date information for reference and practical application to diagnosis and treatment, is an immediately useful book that should be in the library for every family physician, gastroenterologist, and surgeon.

JAMES B. CAREY, M.D.
Minneapolis

Radiation Protection: Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1965)

by PERGAMON PRESS, *New York, 27 pp., 1966.*

This book represents the most recent report of the commission and is a short monograph setting forth their recommendations as of 1965. This commission has dealt with the fundamental principles of radiation protection and its recommendations are expected to serve as guidelines, but not specific rules, for those in various countries who have the responsibility of establishing standards and regulations. For this reason, I believe this book would be most suitable for reference purposes for those directly responsible for radiation protection in medicine, industry, and government. This is not intended to be a textbook on radiation physics or radiobiology. However, a rather complete listing of pertinent publications is given at the end of the book.

It is stressed that much remains unknown regarding the effects of radiation and the relationship to total dose and dose rate. For this reason the recommended doses and exposure limits are quite arbitrary. Some changes in terminology are given but are not of great importance.

The book is divided into 6 sections, the first reviewing basic principles such as types of radiation effects and objectives of radiation protection.

This section also sets forth the Quality Factor (QF) for various types of radiation. This QF must be multiplied by the absorbed dose to give the Dose Equivalent (unit is in rems).

The second section sets forth the limits which have been set for exposure from controlled sources with

(Continued on page 14A)



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(Continued from page 11A)

separate categories for those occupationally exposed and for the general public. It is repeatedly stated that these are recommendations and not hard-and-fast rules. It is also emphasized that medical exposures be limited to the minimum amount connected with medical benefit to the patients. Reference is also made to the recommendation for genetic dose limits to the general population to serve as guidelines in planning industrial uses of radiation and radioactivity.

The third section deals with exposures from uncontrolled sources and is of a very general nature.

The fourth section sets forth general guidelines for radiation protection programs, monitoring, record keeping, and such.

The last 2 sections concern the publications and organization of the commission.

ROBERT J. OLSON, M.D.
Williston

Surgical Diseases of the Chest

edited by BRIAN BLADES, M.D., *2d. ed., 687 pp., ill., C. V. Mosby Co., St. Louis, 1966. $25*

Twenty contributing authors, recognized authorities in their individual fields, have joined to produce the second edition of a text superior to most of the modern books covering the subjects of thoracic surgery.

As a multiauthored text, the quality of each chapter does vary according to the style of the individual. However, the subjects are well presented and the goals of the editor, Dr. Brian Blades, are certainly met, namely, "a book useful not only to the surgeon, but also to the medical practitioners and medical students who are interested in what can be accomplished by the surgical treatment of various diseases of the chest."

Reviewing its pages, one is aware of a better attempt at balance of space and time limiting each subject. For example, pleuropulmonary tuberculosis, by far not the ravage that it was even twenty years ago, is concisely but adequately covered in one 24 page chapter, while cardiac and great vessel diseases, a real problem facing Western civilization medicine today, extends 6 chapters covering 240 pages. The surgery for coronary artery disease alone received a separate chapter because of its recent rapid growth and current importance.

The illustrations and drawings are superior and generously offered, a great help. The quality of the paper and print is excellent, lending to tireless reading and making the chest X-ray reproductions of the finest detail and highest quality.

One of the outstanding features is the excellent bibliography which follows each chapter with the references segmented according to the topic covered, offering an individual reference library on any thoracic surgical subject.

The text is dedicated to the memory of Dr. Evarts Graham who performed the first pneumonectomy for lung cancer and is a worthy and helpful addition to the hospital or doctor's library.

MICHAEL F. LYNCH, M.D.
Minneapolis

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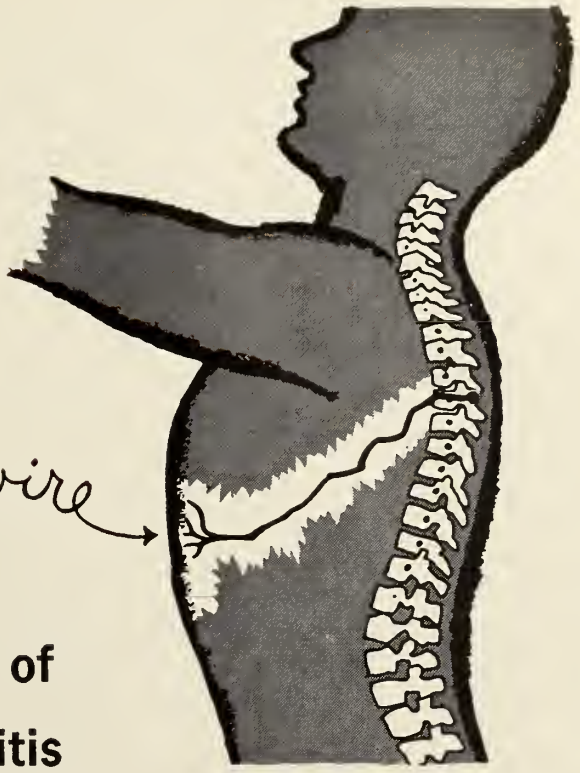
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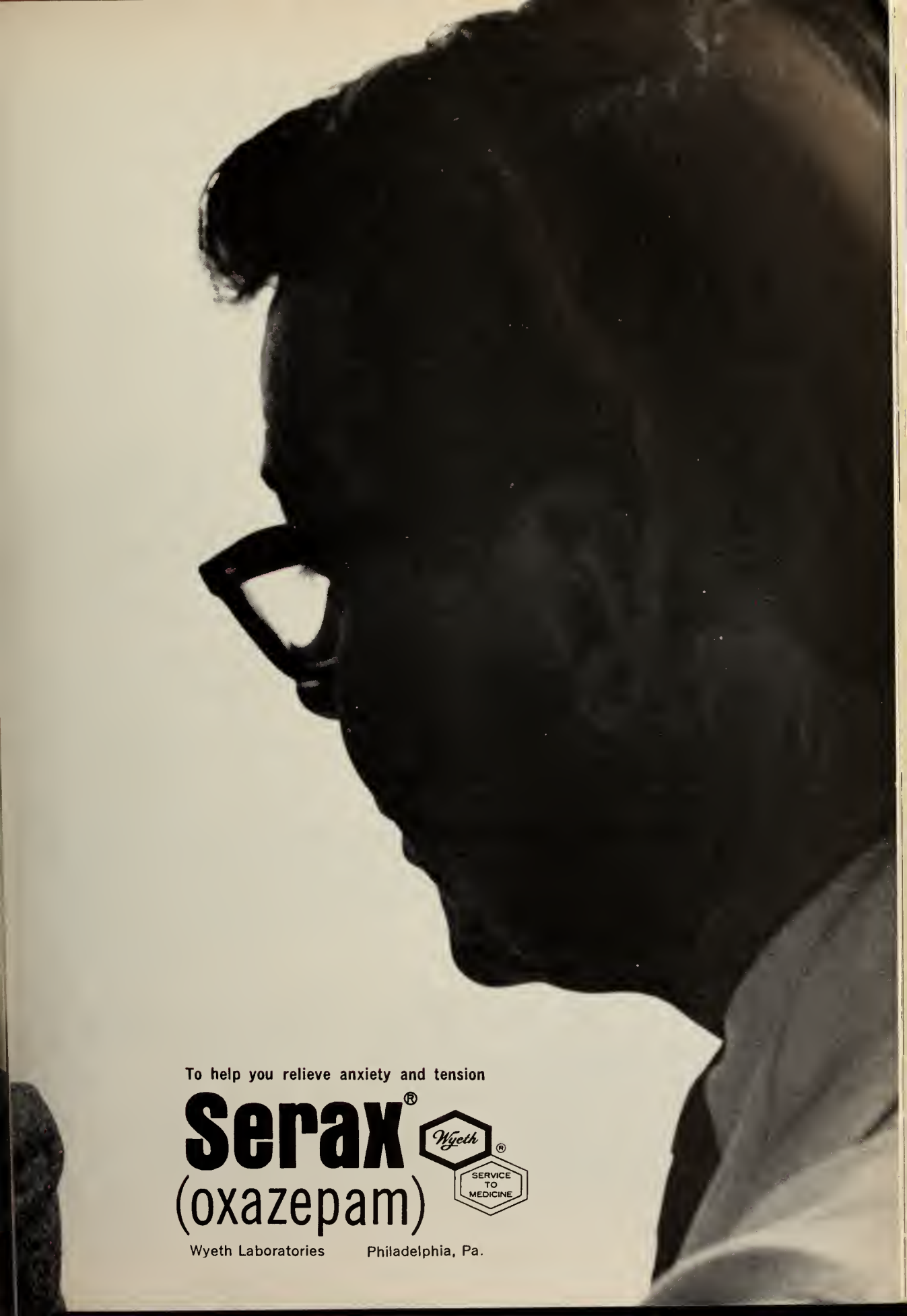
Side Effects: Therapy-interrupting side effects are rare. Transient mild drowsiness is common initially; if persistent, reduce dosage. Dizziness, vertigo and headache have also occurred infrequently; syncope, rarely. Mild paradoxical reactions (excitement, stimulation of affect) are reported in psychiatric patients. Minor diffuse rashes (morbilliform, urticarial and maculopapular) are rare. Nausea, lethargy, edema, slurred speech, tremor and altered libido are rare and generally controllable by dosage reduction. Although rare, leucopenia and hepatic dysfunction including jaundice have been reported during therapy. Periodic blood counts and liver function tests are advised. Ataxia, reported rarely, does not appear related to dose or age.

These side reactions, noted with related compounds, are not yet reported: paradoxical excitation with severe rage reactions, hallucinations, menstrual irregularities, change in EEG pattern, blood dyscrasias (including agranulocytosis), blurred vision, diplopia, incontinence, stupor, disorientation, fever and euphoria.

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News Briefs . . .

North Dakota

DR. KENNETH S. HELENBOLT of Grand Forks was appointed chief of staff of Deaconess Hospital.

* * * *

DR. C. J. GLASPEL of Grafton was presented with a certificate of appreciation by the AMA in recognition of his contributions as a member of the internship review committee for the past eight years.

* * * *

DR. RONALD BROWN, formerly of Bowman, joined the staff of the Ellendale Clinic.

* * * *

DR. CHARLES MAYO II addressed the medical profession and the North Dakota Cancer Society volunteer organization at a dinner meeting in Fargo.

* * * *

DR. E. C. SMART of England will join the Tioga Hospital medical staff in October.

* * * *

DR. JAMES R. AMOS, North Dakota state health officer, was cited by United States Surgeon General William H. Stewart for his leadership and support of an effective injury control program in North Dakota.

* * * *

REELECTED OFFICERS of the North Dakota Tuberculosis and Respiratory Disease Association were Drs. Warren Lee and Robert R. Kling of Bismarck and Dr. Herbert J. Wilson of New Town.

* * * *

DR. A. F. SAMUELSON of Bismarck was named president of the North Dakota Public Health Association.

* * * *

DR. VINCENTE VERZOSA, formerly of the Philippine Islands, joined the Mohall Medical Clinic and the Renville Bottineau Memorial Hospital.

* * * *

DR. ROBERT J. ULMER of Fargo was a delegate to the annual meeting of the American Society of Internal Medicine in San Francisco.

Minnesota

APPOINTMENT of 7 new staff consultants, 3 associate consultants, and 2 chief resident associates in general surgery was announced by the Mayo Clinic, Rochester. The new staff consultants are Drs. Wiley Mitchell Sams, Jr., dermatology; William Douglas, internal medicine; Gerald Gronert, anesthesiology; Jurgen Ludwig, experimental and anatomic pathology; Cameron Strong, kidney and electrolyte research laboratory; James Trautmann, ophthalmology; and William Wharton, occupational medicine. The new associate consultants are Drs. Mark Cloutier, pediatrics; Leroy Dart, Jr., neurosurgery; and

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NEWS BRIEFS

Robert Nichols, neurology. The new chief resident associates in surgery are Drs. William Shamblin and Hal Houston.

* * * *

DR. ELLEN Z. FIFER, formerly health officer for Minneapolis suburbs, was named director of the section of health and rehabilitation planning in the State Planning Agency.

* * * *

DR. W. H. J. SUMMERSKILL of the Mayo Clinic, Rochester, was named a fellow in the Royal College of Physicians, London.

* * * *

DR. L. H. KLEFSTAD of Greenbush will spend six weeks this summer in Madagascar, where he will relieve a mission doctor at the Manambara Hospital in Fort Dauphin.

* * * *

DR. RICHARD MAGRAW, professor of medicine and director of the comprehensive clinic program at the University of Minnesota, spoke at the annual meeting of the American Society of Internal Medicine at San Francisco.

* * * *

DR. RAYMOND D. A. PETERSON, formerly of Minneapolis, was named director of research of the La Rabida-University of Chicago Institute, a medical research organization devoted to the care and study of children with chronic and life-threatening illnesses.

* * * *

DR. ROLAND LARTER, formerly of Lancaster and now completing his internship at Cook County Hospital, Chicago, will set up a practice in the Karlstad Hospital clinic.

* * * *

DR. J. B. BEUNING was appointed Stearns County physician to replace DR. CHARLES F. BRIGHAM, JR., who resigned.

* * * *

DR. HOWARD WONG of Virginia was certified as a diplomate of the American Board of Obstetrics and Gynecology.

* * * *

DRS. D. M. ODLAND and PAUL RUD of Luverne have opened offices in their new Medical Center building.

* * * *

CLINICAL FELLOWSHIPS totaling $45,600 from the American Cancer Society were awarded to the following members of the department of surgery of the University of Minnesota: Drs. John C. Aust, John P. Delaney, Virgil T. Fallon, Arnold S. Leonard, Carl R. McKinley, Allen W. Moberg, and George A. Sarosi.

* * * *

DR. JOHN S. NAJARIAN, who will become head of the surgery department of the University of Minnesota on July 1, serves on a confidential Presidential commission studying the situations of kidney patients facing death for lack of artificial treatment or a transplant. Dr. Najarian now heads a kidney transplant team at the University of California, San Francisco.

* * * *

DR. LEONARD O. BRADLEY of Winnipeg was named president of the Minneapolis Medical Center, Inc., set up by Mount Sinai, Northwestern, and Minneapolis Children's hospitals and the American Rehabilitation Foundation (Kenny Rehabilitation Institute) to establish joint programs and share some facilities.

* * * *

NEW MANKATO CLINIC officers include Dr. Fred D. Roth, board president; Dr. Donald B. Swenson, vice-president;

(Continued on page 20A)

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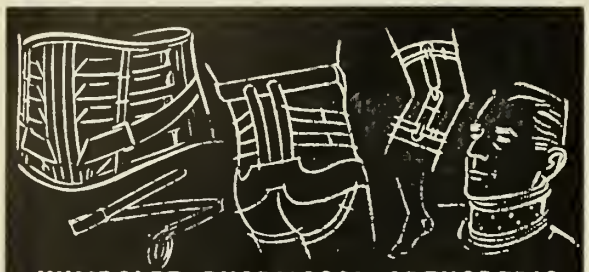
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NEWS BRIEFS

(Continued from page 19A)

Dr. Claire J. A. Strobel, secretary; and Dr. Hobert J. Setzer, Jr., treasurer.

* * * *

DR. HERB L. HUFFINGTON of Waterville, a member of the University of Minnesota Board of Regents, spoke at a dinner meeting of the Southwestern Minnesota Medical Society and Auxiliary.

* * * *

DR. C. GORDON WATSON of Richfield took office as chief of staff at Fairview-Southdale Hospital.

* * * *

DR. EDWARD JORGENSEN, formerly of Duluth, joined the Fergus Falls Clinic and established referral relations with the Park Region Medical Center.

* * * *

DR. WILLIAM C. BERNSTEIN, clinical professor of surgery and director of the proctology division of the University of Minnesota, was honored with the 18th annual Humanitarian Award of the Mount Zion Temple Brotherhood, St. Paul.

* * * *

DR. and MRS. A. F. SETHER of Ruthton were honored by the community for thirty years' service to Ruthton and the surrounding area.

* * * *

DR. C. W. RUCKER, former head of ophthalmology at the Mayo Clinic, Rochester, retired after thirty years on the clinic staff.

* * * *

DR. ARTHUR B. HUNT, former head of a section of obstetrics and gynecology at the Mayo Clinic, Rochester, retired after thirty-two years on the clinic staff.

* * * *

DR. LEONARD AARO of the Mayo Clinic was elected president of the Rochester School Board.

* * * *

DR. WERNER KAESE of Rochester was one of 123 physicians from the United States and foreign countries who attended an intensive postdoctoral course in obstetrics and gynecology at the Boston Hospital for Women.

* * * *

DR. EDWARD CIRIACY of Ely is president of the Minnesota Academy of General Practice.

South Dakota

DR. SEAN O'SULLIVAN, formerly of Ireland, began practice at the Medical Arts Clinic in Redfield.

* * * *

DR. BERNARD HERZOG, formerly of Sioux Falls and now stationed at the United States Naval Hospital in Pensacola, Fla., will join the Milbank Clinic in July.

* * * *

DR. JOHN ELSTON of Rapid City was reelected chairman of the Pennington County Board of Health and will continue to serve as county health officer.

* * * *

DR. ANTHONY PETRES of Salem was elected to active membership in the American Academy of General Practice.

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NEWS BRIEFS

Deaths . . .

DR. ARTHUR C. KERKHOF, 63, Minneapolis, died May 3. A prominent cardiologist, he was a member of the national Board of Directors of the American Heart Association and chairman of the American Therapeutic Committee on Education.

* * * *

DR. HAROLD J. ROTHSCHILD, 83, St. Paul eye, ear, nose, and throat specialist, died April 20. His entire medical practice was in the Twin Cities area.

* * * *

DR. WILLIAM T. WALSH, 56, Minneapolis, died April 6. He practiced medicine in Minneapolis and was chief of staff of St. Barnabas Hospital in 1956.

* * * *

DR. CARL G. SWENDSEEN, 72, Pompano Beach, Fla., died April 9. He was formerly a prominent ear, nose, and throat specialist and chief of staff of Swedish Hospital in Minneapolis.

* * * *

DR. FRANK J. ELIAS, 74, Duluth, died April 7. He was a partner in the Morgan Park Medical Center, a past president of the Minnesota Medical Society, and the recipient of the society's Distinguished Service Medal in 1954.

* * * *

DR. CHRISTIAN A. FJELDSTAD, 83, Minneapolis, died April 21. He had practiced ophthalmology in Minneapolis for forty-seven years.

* * * *

DR. ELWYN R. BRAY, St. Paul surgeon, died March 19. He was a 1901 graduate of the University of Minnesota medical school and trained in eye specialization in London and Vienna.

SACRED HEART, MINNESOTA has been without a doctor since 1961. General practitioner will find suitable building in 700 pop. village, modern hospital 10 miles away; progressive, stable community in west central part of state. Good school, churches. Good family environment. Call or write Rev. Robert Bardy, Telephone 765-2547, Sacred Heart, Minn.

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GENERAL PRACTITIONER wanted to join well established practitioner. New clinic building, 90 miles east of St. Paul. City of 14,000. Early partnership with full hospital facilities. Ideal location. L. W. Picotte, M.D., Chippewa Falls, Wisconsin.

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1 Sherman, L. F.: Minnesota Med 48,605. Mo;, 1965. 2 Sherman, L. F. Personal Communication.

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Each 60 cc. unit contains:

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Description: CORTENEMA Hydrocortisone Retention Enema is a completely disposable, single-dose unit consisting of a flexible plastic container with a lubricated applicator tip for rectal administration. It is designed to permit self-administration and, therefore, it is suitable for both out-patient and hospital use. It also may be diluted with isotonic saline for administration by intrarectal drip.

CORTENEMA consists of a suspension of partially solubilized hydrocortisone alcohol in an aqueous vehicle. Thus, it provides the potent anti-inflammatory effect characteristic of the corticosteroids. Because hydrocortisone alcohol is absorbed to some extent from the colon, CORTENEMA provides both topical and systemic actions.

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Rectal steroid therapy brings about prompt symptomatic and proctologic remissions in a significant proportion of patients, but it apparently does not alter the recurrent and progressive nature of ulcerative colitis. Therefore, this form of treatment is not recommended for prophylaxis during periods of remission.

Contraindications: Local contraindications to the use of intrarectal steroids include obstruction, abscess, perforation, peritonitis, fresh intestinal anastomoses, extensive fistulas and sinus tracts. Tuberculosis (active, latent or questionably healed), ocular herpes simplex, and acute psychosis are usually considered absolute contraindications to the use of corticosteroids.

Relative contraindications include active peptic ulcer, acute glomerulonephritis, myasthenia gravis, osteoporosis, diverticulitis, thrombophlebitis, psychic disturbances, pregnancy, diabetes, hyperthyroidism, acute coronary disease, hypertension, limited cardiac reserve, and local or systemic infections, including fungal or exanthematous diseases. Where these conditions exist, the expected benefits from CORTENEMA must be weighed against the risks involved in its use.

If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

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DISPOSABLE UNIT

FOR RECTAL USE ONLY

Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

How Supplied: CORTENEMA Hydrocortisone Retention Enema is supplied in boxes of seven single-dose units.

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References: 1. Musser, Ruth D., and Shubkagel, Betty L.: Pharmacology and Therapeutics, ed. 3, New York, Macmillan Company, 1965, p. 363. **2.** Council on Drugs, American Medical Association: New Drugs 1965, Chicago, American Medical Association, 1965, p. 157. **3.** Modell, Walter (Ed.): Drugs in Current Use 1966, New York, Springer Publishing Company, Inc., 1966, p. 77.

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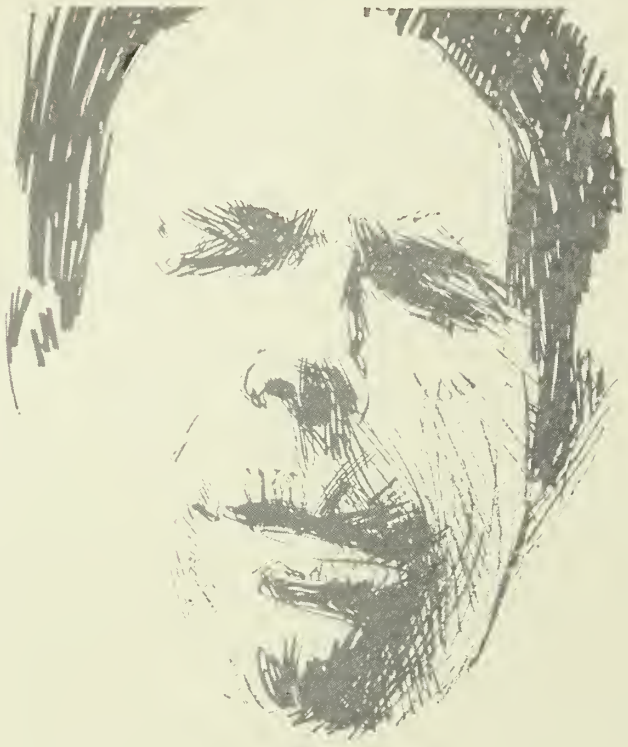
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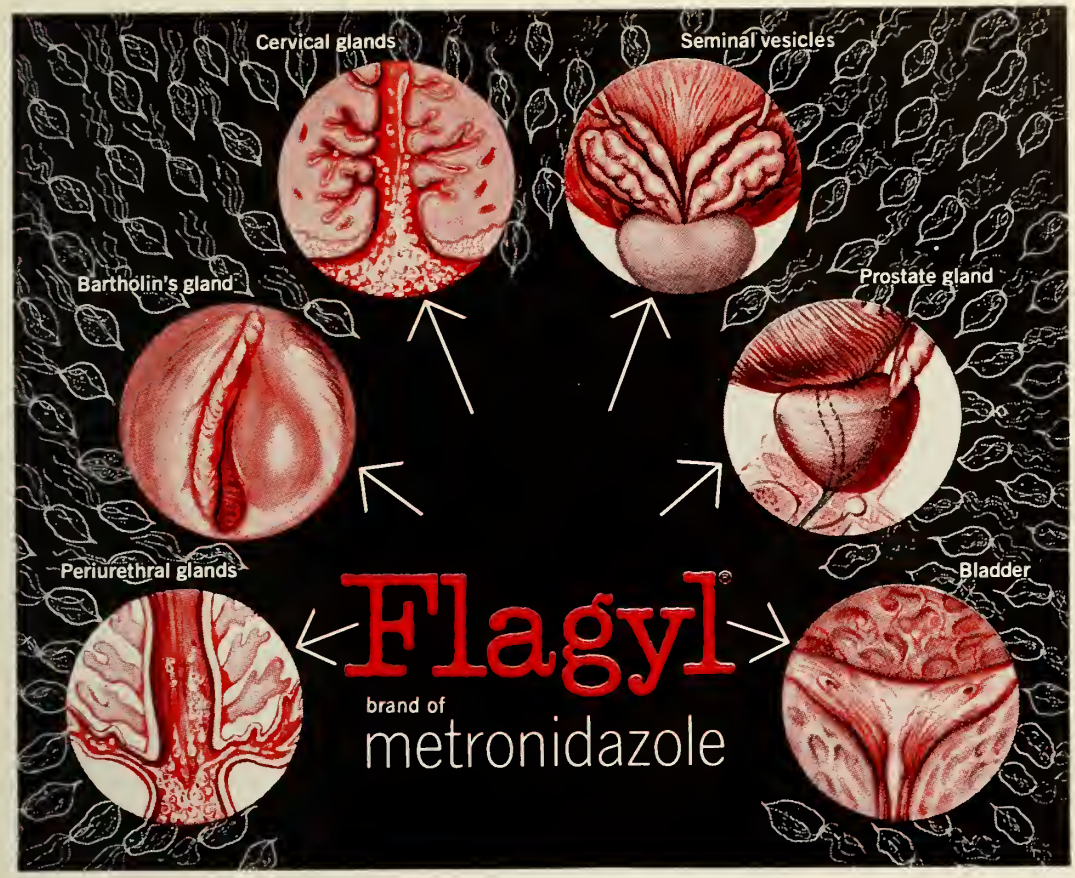
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CONTENTS

St. Paul–Ramsey Hospital Symposium
A fitting tribute—a challenge . . . 229
D. H. Peterson, M.D.
Acute Rheumatic Fever: A New Look . 230
Russell V. Lucas, Jr., M.D.
What About the Small Bowel? . . 236
Milton R. Gilchrist, M.D.
Vesicoureteral Reflux: A Brief Review . 238
C. D. Creevy, M.D.
Chondritis as a Postoperative Complication . 247
F. John Lewis, M.D.
The Objective Evaluation of Auditory Function . . . 251
Sister Victorine Long, M.S., M.T., (ASCP), L. R. Boies, Jr., M.D., J. A. Hilger, M.D., and N. A. Roth
Radiotherapy: Art and Science 1965 . . . 256
G. J. D'Angio, M.D.
A Neglected Roentgen Diagnostic Feature of Ruptured Aneurysms of the Abdominal Aorta 259
I. Nik Nevin

A Transsexual Research Project at the University of Minnesota Medical School . . 262
Donald W. Hastings, M.D., and John A. Blum, M.D.
Mediastinal Abscess of Subdiaphragmatic Origin 265
Warren R. Nickerson, M.D., Floyd E. Weaver, M.D., and William E. Neville, M.D.
A Promising Procedure for the Treatment of Urinary Incontinence in the Male . 268
John H. Hoskins, M.D., and George T. Mellinger, M.D.
John A. Anderson, M.D. 270
Lewis W. Wannamaker, M.D.
Book Reviews 11A
News Briefs 19A

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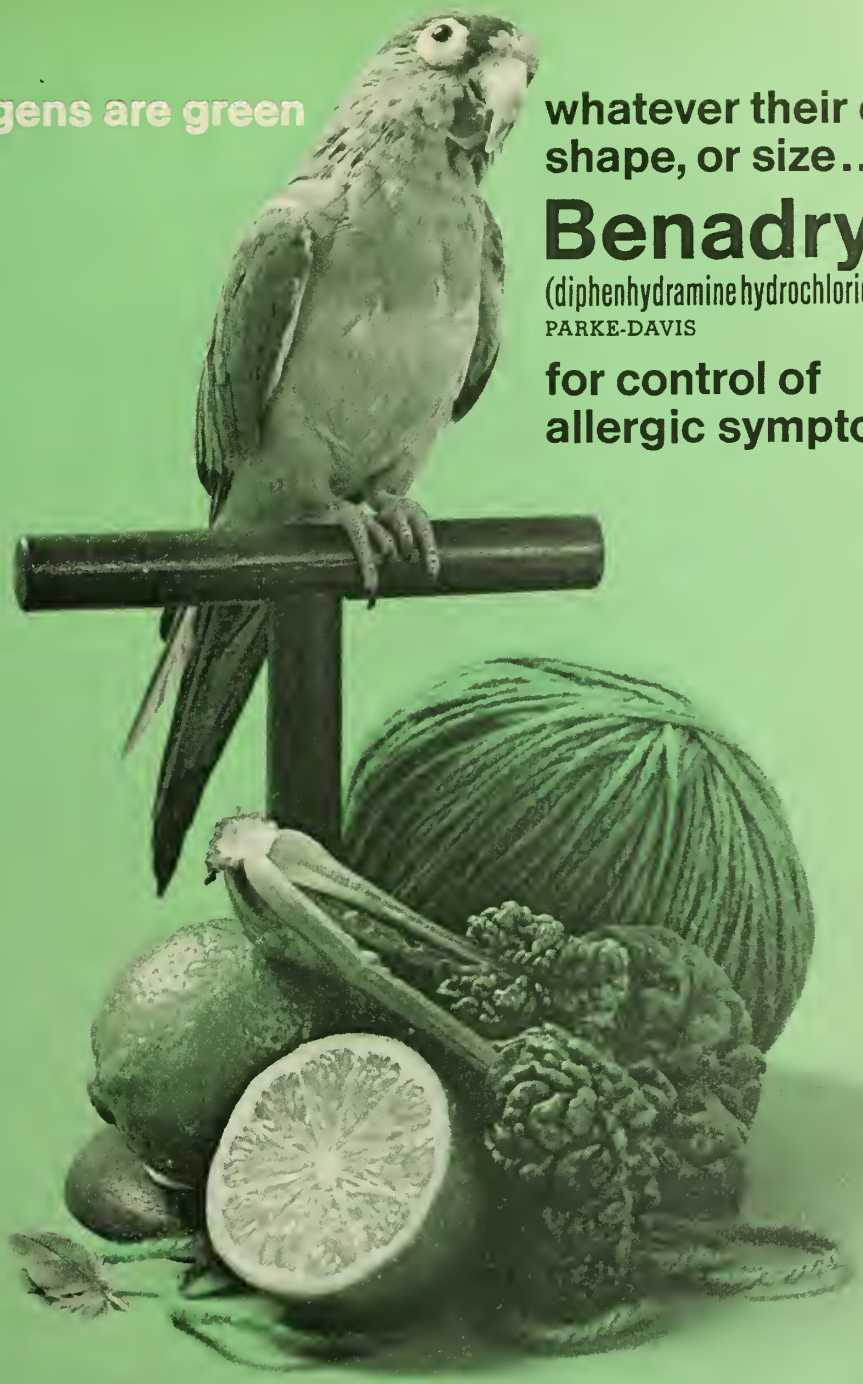
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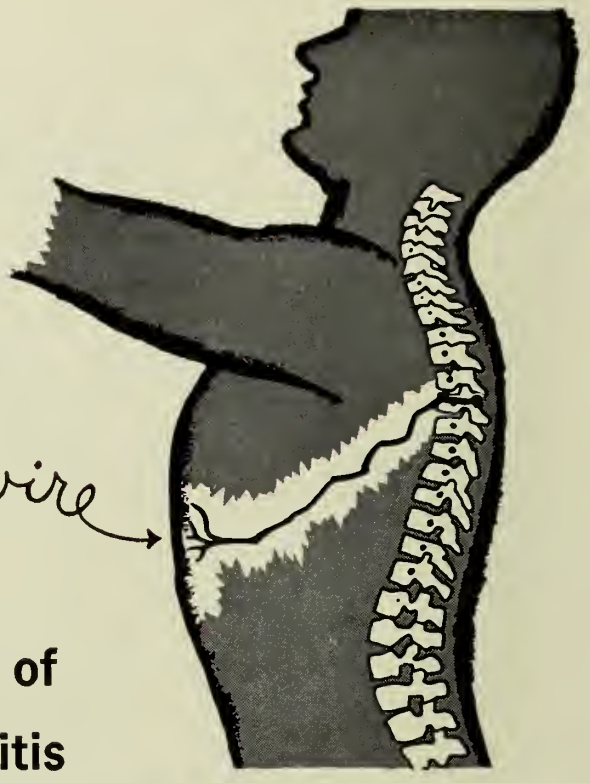
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St. Paul-Ramsey Hospital Symposium

A fitting tribute—a challenge

The articles in this edition of the *Journal-Lancet* have been collected from an Inaugural Scientific Program held at the St. Paul–Ramsey Hospital, St. Paul, Minn., in November 1965. The time of the program was shortly after the hospital staff and the patients of Ancker Hospital had moved to its new location, the St. Paul–Ramsey Hospital.

The old Ancker Hospital was rich in traditions and memories. It had served the community for ninety-two years in the care of patients as well as the training of doctors. Since the beginning of the intern program in 1887, approximately 1,500 interns and residents had received training in this old hospital. Since 1945, there has been an active affiliation with the University of Minnesota in the training of residents and medical students. In this background, it was fitting and right to acknowledge the role that Ancker Hospital had played in patient care and medical education. The excellence of the job was attested to by the number of medical men who responded to the homecoming call from their alma mater to return and participate, either directly or indirectly, in this program. The number and variations of the subjects discussed was an indication of the great variety of interests that these men have been involved in in their practice and teaching. The program indeed was a fitting tribute to a job well done.

On the other hand, the program was a fitting challenge to a new institution and its medical staff. While the goals of the hospital in the care of patients and in its community activities are quickly evaluated, the goal of medical education and research is one which time alone will tell. The goals of the present hospital must be set higher not because of any unusual abilities of the medical educators of today but because of the impetus and precedent which have been set by the medical men of yesterday. A fitting tribute has been made. A challenge has been accepted.

D. H. PETERSON, M.D., *chief of staff*



City and County Hospital, 1884 (Left)
St. Paul–Ramsey Hospital, 1967 (Below)



Ellerbe Architects

Acute Rheumatic Fever: A New Look

RUSSELL V. LUCAS, JR., M.D.
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THE INCIDENCE of acute rheumatic fever has dramatically decreased in the last thirty years. This clinical impression is held by physicians throughout the United States and western Europe and is supported by the available statistical evidence. A summary by Collins[1] of 6 surveys in this country spanning the years 1928 to 1943 revealed an acute rheumatic fever attack rate of 100 to 120 per 100,000 children. A more recent survey by Stamler[2] in 1962 estimated the attack rate in Chicago children to be 50 per 100,000. Further, the prevalence of rheumatic heart disease has shown a dramatic decrease in school children,[3–7] in military recruits,[8] and in college students.[9]

Fully as important as the decrease in incidence in rheumatic fever is the decline in its severity. The incidence of chorea, subcutaneous nodules, and erythema marginatum is lower. The severity of carditis has undergone a dramatic diminution.[10] Death rates from acute rheumatic fever in all age groups have fallen from 16.8 per 100,000 in 1910 to 0.4 per 100,000 in 1960.[11]

In summary, modern rheumatic fever is different. There is much less of it and the cases we see are considerably more benign.

Why the change? While its precise pathogenesis is unknown, rheumatic fever is a sequel to group A, beta-hemolytic streptococcal infection. We have no evidence that the streptococcus has changed its characteristics. Attack rates of rheumatic fever following epidemic streptococcal outbreaks have not changed. An attack rate of 3.6% was observed in Denmark in 1926,[12] and a comparable rate was noted by Rammelkamp and his co-workers among World War II recruits.[13] However, the incidence of rheumatic fever following endemic or sporadic streptococcal infection is strikingly less than that seen after epidemic infections.[14,15] Epidemic outbreaks of streptococcal disease are fewer now. This accounts for the decrease in acute rheumatic fever, even though the incidence of sporadic streptococcal infection remains high.

The major factor responsible for the decrease in epidemic streptococcal disease appears to be the improvement of social conditions, particularly the alleviation of crowded living accomplished by migration to the suburbs, by slum clearance, and by municipal redevelopment. Thus, the decline in acute rheumatic fever is a social triumph, not a medical one.

Penicillin has contributed little to the decreased incidence of acute rheumatic fever. The trend was clearly established before the antibiotic era. The chief role of penicillin to date has been the prevention of recurrence of rheumatic fever by the introduction of continuous prophylaxis and, thus, a reduction in severe rheumatic heart disease. Its effectiveness in preventing the initial attack of rheumatic fever through treatment of streptococcal infections has been disappointing, except in special populations such as military recruits.

The diagnosis, treatment, and prevention of acute rheumatic fever will be considered in relation to today's changing pattern of the disease.

DIAGNOSIS

Acute rheumatic fever remains a disease without a single pathognomonic sign, symptom, or laboratory test. In 1944, T. Duckett Jones[16] outlined criteria for the diagnosis of acute rheumatic fever. These were initially modified by a committee of the American Heart Association in 1955. In 1965, the Jones Criteria were again revised by a committee of the American Heart Association (see table).[17]

These criteria are useful to the clinician in making a diagnosis of acute rheumatic fever. The presence of two major criteria or of one major and two minor criteria, plus evidence of preceding streptococcal infection, indicates a high probability of rheumatic fever.

EVIDENCE OF PRECEDING STREPTOCOCCAL INFECTION

The main change in these revised criteria is in the greater stress now placed upon evidence

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of a preceding streptococcal infection. In the absence of this evidence, a diagnosis of acute rheumatic fever should be made with caution.

This is particularly true in the patient with isolated carditis, since myocarditis and pericarditis of viral etiology are increasingly recognized in infants, children, and adults. Other causes of carditis are also better appreciated at the present time.

Thus, the presence of evidence of streptococcal infection supports the diagnosis of rheumatic fever in the patient with carditis, while the absence of such evidence suggests one of the other etiologies.

The most reliable evidence of streptococcal infection is an increased or rising streptococcal antibody titer. The antibody levels are elevated in the first two months following a streptococcal infection; if detection of rheumatic fever is delayed longer than this, the antibody titers may not be significantly elevated. Antistreptolysin O (ASO) is the titer most widely used and best standardized. A level of 333 units or higher in children over 5 years of age is considered increased. A two-tube rise in titer is significant.

JONES CRITERIA (REVISED)* FOR GUIDANCE IN THE DIAGNOSIS OF RHEUMATIC FEVER

Major manifestations	*Minor manifestations*
Carditis	Clinical
Polyarthritis	Previous rheumatic fever or rheumatic heart disease
Chorea	Arthralgia
Erythema marginatum	Fever
Subcutaneous nodules	Laboratory
	Acute phase reactions: erythrocyte sedimentation rate, C-reactive protein, leukocytes
	Prolonged pulse rate interval

Plus

Supporting evidence of preceding streptococcal infection

- Increased antistreptolysin titer or other streptococcal antibodies
- Positive throat culture for group A streptococcus
- Recent scarlet fever

The presence of two major criteria or of one major and two minor criteria indicates a high probability of the presence of rheumatic fever if supported by evidence of a preceding streptococcal infection. The absence of the latter should make the diagnosis doubtful, except in situations in which rheumatic fever is first discovered after a long latent period from the acute antecedent infection.

*Revised October 1965 by a committee of the American Heart Association[17]

Other antibody tests for the detection of streptococcal infection are antihyaluronidase (ASH), antistreptokinase (ASK), antideoxyribonuclease B (anti-DNAse B), antidiphosphopyridine nucleotidase (anti-DPNase), and antinicotinamide adenine dinucleotidase (anti-NADase). When two or more of these antibody tests are performed within the first two months of onset, almost all cases of rheumatic fever show increased titer in at least one test.[16]

Isolation of group A, beta-hemolytic streptococcus by throat culture is less reliable. Since the infection has been present for some weeks, the organisms are usually present in small numbers and may be absent, particularly if antibiotics have been given. Furthermore, many normal asymptomatic children harbor group A streptococci.

A history of a preceding attack of scarlet fever is an excellent indication of antecedent streptococcal infection.

MAJOR MANIFESTATIONS

Migratory polyarthritis, a characteristic, major manifestation of acute rheumatic fever, is indicated by swelling, redness, heat, tenderness, pain, and limitation of motion in two or more joints. It is almost always migratory. It is important to point out that arthralgia is not a major manifestation of acute rheumatic fever but a minor one.

Chorea consists of purposeless, involuntary, rapid movements, often associated with clumsiness in dressing and eating and with emotional instability. When chorea occurs, it is frequently an isolated and delayed manifestation and usually appears some months after the precipitating streptococcal infection. Evidence of preceding streptococcal disease is often lacking because of the greater duration of the latent period.

Erythema marginatum is a recurrent, pink, evanescent rash with scalloped edges and fading centers. The rash does not occur on the face. It is often brought out by warming, is not pruritic or indurated, and blanches on pressure.

Subcutaneous nodules are firm, painless, pea-sized nodules primarily located over the extensor surfaces of the joints, occiput, and spinous processes. They are nontender and the overlying skin moves freely.

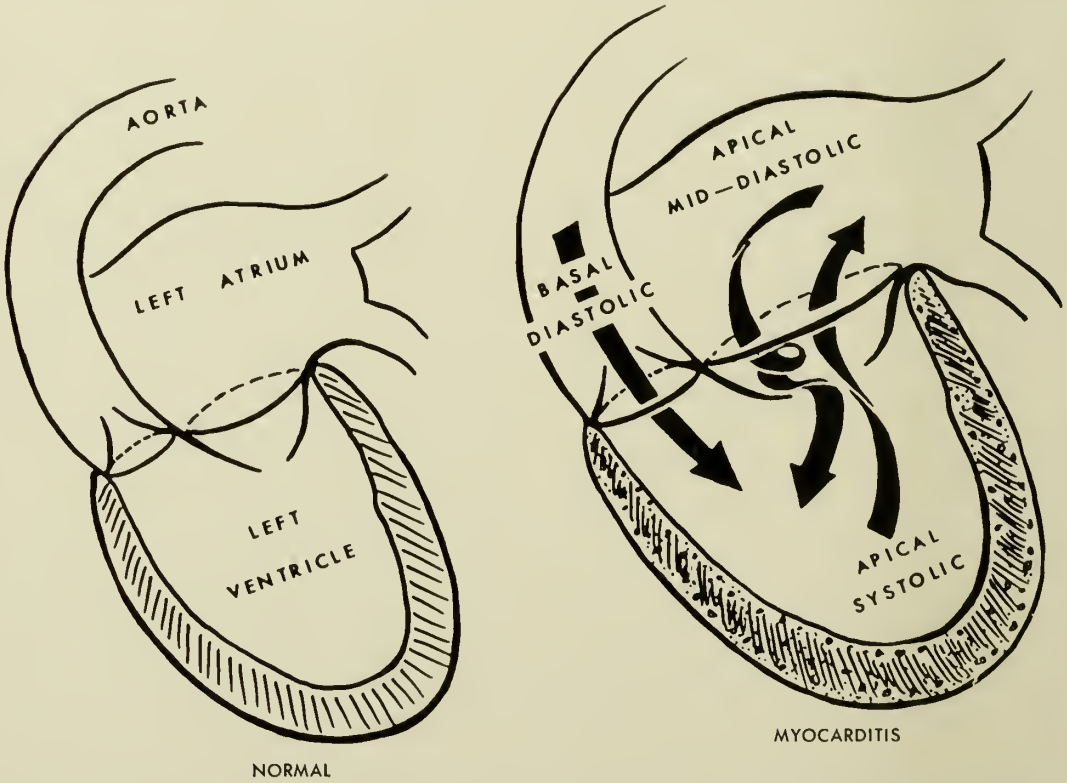
Carditis is the most critical major manifestation. Acute rheumatic fever without carditis is a benign, self-limited disease. The presence of and severity of carditis are the major determinants of the prognosis of rheumatic fever in any given patient. Thus, the diagnosis of carditis is important not only to the establishment of the

presence of acute rheumatic fever but it is of great importance in assessing the prognosis. The carditis of acute rheumatic fever is a pancarditis involving all three layers of the heart. In the acute stage, pericarditis and myocarditis produce the significant signs and symptoms. Initially, endocarditis produces little in the way of signs or symptoms, but it does produce significant scarring of valves and impairment of valve function, effects that usually appear years or decades after the acute attack.

Cardiac murmurs are almost always associated with acute rheumatic carditis. It is important to point out, however, that the murmurs (or other signs of carditis) are in no way specific for rheumatic carditis. Carditis of any etiology may produce precisely the same signs. This suggests that these murmurs are not a result of damage to the valvular endocardium but, rather, are secondary to myocardial damage. In myocarditis of any etiology, impaired ventricular function results. The left ventricle is most affected, since it does the most work. The weakened left ventricle dilates to gain the advantages of greater diastolic length, as defined by Starling's law.

As the left ventricle dilates, the mitral valve ring also enlarges (see figure). Now the valve cusp can no longer guard the mitral valve orifice during systole. Mitral regurgitation occurs and produces an apical, systolic murmur. The murmur is holosystolic, blowing, and high-pitched and is heard in the apical region and transmitted toward the axilla and back. As a consequence of mitral regurgitation, the left atrium dilates. Since the atrial wall is continuous with the posterior leaflet of the mitral valve, dilatation of the left atrium produces tension on and shortening of the posterior leaflet, the mitral orifice is less well guarded, and more regurgitation occurs.

During diastole the increased left atrial blood volume is transferred to the left ventricle. Even though the mitral valve ring is enlarged, the greatly increased amount of blood flowing from left atrium to left ventricle during diastole often results in an apical, middiastolic murmur. This is a low-pitched, short, middiastolic murmur



Schematic representation of the murmurs of carditis. [Left] Normal. [Right] Left ventricular dilatation secondary to myocarditis. The valve rings are also stretched. The normal mitral valve leaflets can no longer guard the mitral orifice, mitral regurgitation occurs, and the apical systolic murmur results. Also as a consequence of mitral regurgitation, the left atrium must return a large volume of blood to the left ventricle during diastole and this produces the apical middiastolic murmur. Dilatation of the aortic valve ring allows aortic regurgitation to which the basal diastolic murmur testifies

that is best heard in the left lateral recumbent position at the apex with the breath held in expiration. This murmur is separate and distinct from the crescendo presystolic murmur indicative of established organic mitral stenosis.

Dilatation of the aortic valve ring, which occurs less often in acute carditis, allows aortic regurgitation that produces a basal diastolic murmur. This murmur is heard early in diastole and is a high-pitched, blowing, decrescendo murmur heard along the left sternal border. The murmur of aortic insufficiency may be intermittent and its detection requires careful auscultation.

Each of these three murmurs is a consequence of dilatation of the left ventricle. The presence of one or more of them is diagnostic of carditis (but not necessarily rheumatic carditis). They are easily distinguished from the functional murmurs frequently heard in children and from those produced by congenital cardiac anomalies, except congenital mitral insufficiency.

Pericarditis manifested by friction rub, pericardial effusion, or definite electrocardiographic changes is also diagnostic of carditis. A diagnosis of carditis can be established when unequivocal cardiac enlargement exists in a patient without history of previous rheumatic fever or other cardiac defect. Moreover, an obvious increase in cardiac size in a patient with a past history of rheumatic heart disease is evidence of a recurrent, acute episode of rheumatic fever.

Finally, congestive cardiac failure in a child or young adult, in the absence of any other cause of congestive heart failure, is highly suggestive of carditis.

MINOR MANIFESTATIONS

These clinical features occur often in acute rheumatic fever, but they so frequently occur in other disease states that their diagnostic significance is minor. Their presence is of value only as supportive evidence of rheumatic fever when at least one major manifestation is clearly present. They have no diagnostic significance in the absence of a major manifestation (see table).

OVERDIAGNOSIS OF RHEUMATIC FEVER

The presence of acute rheumatic fever is rarely overlooked by the conscientious physician. On the other hand, overdiagnosis is common and results in significant morbidity. The diagnosis should be approached conservatively, and the presence of unequivocal, major clinical manifestations should be insisted upon. In particular, the clinician should resist the temptation to administer steroids or salicylates before the signs and symptoms are unmistakable. Should this be done, an ill-defined picture is left that is only presumptively rheumatic fever, and subsequent intelligent management is most difficult.

Grossman and Athreya[18] have tabulated the sources of error in 455 patients mistakenly diagnosed as having acute rheumatic fever. The most common error, occurring in well over half the patients, was the misinterpretation of a functional cardiac murmur for one of the specific murmurs of carditis. Arthralgia was mistaken for migratory polyarthritis in about one-fourth of cases. An erroneous diagnosis of streptococcal infection occurred in roughly 20% of the patients. Other errors included the misinterpretation of fever, easy fatigability, precordial pain, and abnormal movements. Personal experience has shown that the most common diagnostic error is misinterpretation of innocent heart murmurs and of vague muscle pains.

TREATMENT OF ACUTE RHEUMATIC FEVER

Eradicate the streptococcus. This is the first principle of treatment. The patient with acute rheumatic fever must be assumed to have residual beta-hemolytic streptococci in the nose or throat, even though cultures are negative. Eradication of these organisms necessitates adequate penicillin blood levels for ten days. Appropriate treatment is 0.6 to 1.2 million units of benzathine penicillin G in one injection. Adequate blood levels may also be obtained by oral penicillin G in a dosage of 200,000 units three or four times daily for ten days. In the event that penicillin cannot be used, a ten-day course of erythromycin is satisfactory. Sulfonamides do not eradicate streptococci.

Relieve joint pain and fever. For the symptomatic relief of joint pain and fever, aspirin in a dosage of 1 gr. per pound per day in six divided doses is almost always efficacious. The dose should be reduced by one-third after forty-eight hours. The majority of patients will tolerate this dosage, but occasionally signs of toxicity are produced. In particular, the physician should be alert for hyperpnea.

Treat carditis. No specific treatment exists for rheumatic carditis. Aspirin and steroids both reduce the acute inflammatory response, hasten the disappearance of pericarditis, and probably have a beneficial effect on the myocarditis. There is agreement that when life is threatened the steroid drugs are of more benefit and their use is encouraged. Many physicians also consider the presence of congestive cardiac failure an indi-

cation for the use of steroids. Steroids are started at a relatively high dose, such as 2 to 4 mg. per pound per day of cortisone in divided doses or prednisone at one-fourth this dose. When symptoms are controlled, the dose is dropped to the minimum amount that will maintain symptomatic relief.

Congestive heart failure should be treated in the usual way, using a digitalis preparation, diuretics, salt restriction, and oxygen. Digoxin is a preferred drug, since it has a relatively rapid onset of action and a relatively rapid rate of excretion.

Since neither steroids nor salicylates shorten the course of rheumatic fever, duration of therapy must cover the expected course of the attack. About 80% of the attacks of acute rheumatic fever will subside clinically within six weeks and about 90% within twelve weeks.[19] Few relapses thus occur if treatment is maintained for twelve weeks. It is important to gradually reduce the steroid dosage over a period of about two weeks before discontinuation to prevent a state of temporary adrenal insufficiency.

The erythrocyte sedimentation rate (ESR) and the C-reactive protein test (CRP) are useful in assessing the effectiveness of anti-inflammatory therapy as well as in detecting exacerbations of rheumatic activity. These two tests are elevated during the period of rheumatic activity and normal during adequate suppression or remission. The CRP reacts more quickly to clinical change than does the ESR and is not lowered by congestive heart failure as the ESR often is.

Finally, the patient with carditis should be placed on bed rest. Bed rest should not be enforced in acute rheumatic fever in the absence of carditis. The children with painful joints place themselves at rest, and when the joint symptoms are controlled, there is no longer any need to enforce bed rest. It is well to recall that the rationale for bed rest is reduction of cardiac output and, thus, rest for the diseased heart. The cardiac work involved in the use of a bedpan and in resisting bed rest is often greater than that which would be entailed by bathroom privileges and a program of moderate activity. The usual child has the ability to maintain his activity at an optimal level. Moreover, strictly enforced bed rest for a prolonged period of time may result in severe muscle wasting and weakness, lethargy, osteoporosis, joint stiffness, or contractures. After prolonged periods of bed rest, increase in activity often results in joint and muscle pain, malaise, and elevated pulse rate and these may be mistaken for an exacerbation of acute rheumatic fever. This problem may be avoided by allowing modified activity early.

Prevention of permanent valve damage. Almost uniform agreement exists now that steroids do not prevent permanent valve damage due to acute rheumatic fever. The United Kingdom and United States Cooperative Clinical Trial has just published its ten-year report.[20] In this well-controlled study of 497 children, the relative merits of ACTH, cortisone, and aspirin in the treatment of acute rheumatic fever were assessed. Evaluations at one, five, and ten years revealed no evidence that, on the treatment schedule used in the study, the prognosis has been influenced by one treatment over another.

The major criticism of this study was the relatively short duration (six weeks) and the low dosage of steroids. To determine whether large doses of steroids over a longer period of time were superior to salicylates, the Combined Rheumatic Fever Study Group was formed and conducted a controlled study in 55 patients utilizing large doses of steroids for twelve weeks.[21] They were unable to show that prednisone at high dosages for twelve weeks had any superiority over aspirin.

On the basis of these two studies, most authorities accept the position that steroids are not significantly better than aspirin in the prevention of residual valve damage in acute rheumatic fever. Thus, when steroids are used, they are used for control of acute symptoms and their dosage and duration should be that minimum necessary to achieve this goal.

PREVENTION OF RHEUMATIC FEVER

Prevention of recurrent rheumatic fever in rheumatic individuals. The rheumatic patient shows an increased incidence of rheumatic fever following a streptococcal infection, but recurrence can be avoided if streptococcal infections are prevented. Since many streptococcal infections occur without producing clinical manifestations, prevention depends on continuous penicillin prophylaxis rather than on recognition and treatment of acute attacks of streptococcal disease.

All patients with a well-documented history of rheumatic fever and all patients with rheumatic heart disease should receive prophylaxis. The safest policy is to continue prophylaxis for life. A minimum requirement must be to at least age twenty-five or for five years after the last attack of acute rheumatic fever in the adult.

The most reliable results have been obtained with intramuscular benzathine penicillin G, 1.2 million units per month.[22] Despite its better

reliability, patient acceptance of this mode of treatment is poor because of the discomfort at the injection site, which may last several days. Oral penicillin and oral sulfonamide therapy are equally effective in preventing streptococcal infection. However, the success of oral medication depends on regular ingestion of the medication. It is a physician's responsibility to repeatedly instruct patients on the need for regular ingestion. Dosage of sulfadiazine is 1 gm. daily for patients over 60 lb. and 0.5 gm. for a patient under 60 lb. Penicillin G is given in a dosage of 200,000 units once or twice daily. Twice daily is probably more effective.[22]

Prevention of the initial attack of rheumatic fever. In theory, recognition and treatment of a streptococcal infection within nine days of its onset will prevent the occurrence of the initial attack of rheumatic fever. In practice, however, about half the streptococcal infections which occur are likely to escape detection because they are asymptomatic or the symptoms are atypical. In the other half, clinical manifestations are sufficient to suggest the possibility of streptococcal infection, but bacteriologic support by throat culture is necessary for confirmation. Recent studies have investigated the significance of group A, beta-hemolytic streptococci isolated from children with uncomplicated nasopharyngitis. Top and Wannamaker[23] found that 34% of 259 children with nasopharyngitis harbored group A, beta-hemolytic streptococci. More than half of these were not typable and were presumed to be avirulent. Only 14% of the patients who harbored streptococci had a two-tube rise in anti-DNAse B. Their conclusion was that most of these patients with nasopharyngitis and with group A, beta-hemolytic streptococcus cultured from the nasopharynx did not have streptococcal disease but, rather, were carriers with unrelated, coincident viral nasopharyngitis or they had had streptococcal infection in the more distant past.

The foregoing should not be interpreted to mean that throat cultures are of no value or that intelligent management of the patient with sore throat is not possible. Stollerman and coworkers[24] have outlined a practical approach for the physician handling a patient with pharyngitis.

1] A negative throat culture virtually excludes the need for antibiotic therapy (excepting rare circumstances, such as diphtheria).

2] Negative throat cultures in a succession of patients with exudative pharyngitis strongly suggest the outbreak of a viral infection capable of mimicking streptococcal pharyngitis. The physician can confidently omit antibiotic therapy in these patients.

3] The patient with exudative pharyngitis and a positive throat culture requires antibiotic therapy.

4] If several of these cases occur in succession, the physician can identify an incipient epidemic of streptococcal disease. This situation requires treatment of the patient, culture (and if necessary, treatment) of his intimate contacts, and prompt culture of all suspect patients in the physician's practice.

It is well to recall that epidemic streptococcal infection is much more likely to result in rheumatic fever than is sporadic infection. Thus, recognition of the incipient streptococcal epidemic and its subsequent interruption offers the greatest opportunity for the further reduction in the incidence of acute rheumatic fever. At present, this requires that the private practitioner continuously monitor by throat culture at least a significant sample of his practice.

The ultimate in control over the streptococcal epidemics in a community will be achieved when the results of each physician's throat cultures are quickly correlated, combined with virulence testing and antibody titers, and the results returned to the medical community for action. This approach requires only the cooperation of physicians and the local health departments plus an appropriate organizational structure, and it appears close to fruition in some areas.

SUMMARY

The incidence of acute rheumatic fever is decreasing and its severity is lessening. The main reason for these changes appears to be a decrease in epidemic streptococcal disease, the result of better social conditions.

The recent revision (October 1965) of the Jones criteria for guidance in the diagnosis of acute rheumatic fever is reviewed. The greatest change is the stress placed on evidence of antecedent streptococcal infection.

An evaluation is made of the treatment of acute rheumatic fever and the place of steroids in therapy.

Prevention of the initial attack of rheumatic fever can be accomplished through control of streptococcal infection. The methods of recognition and control of streptococcal disease are enumerated.

(Continued on page 258)

What About the Small Bowel?

MILTON R. GILCHRIST, M.D.

We examine the esophagus, stomach, duodenum, and colon with great thoroughness—but what about the small bowel? Abnormalities of the small intestine, including tumors, may be clinically unsuspected. Signs and symptoms may be so vague that a small bowel study is not requested by the referring physician. Most institutions do not carry out a routine examination of the small bowel following a study of the stomach and duodenum. At 1 local hospital over a six-month period there were 2,340 examinations of the esophagus, stomach, duodenum or colon, but only 130 examinations of the small bowel.

In a Lancet[1] series of 190 small bowel examinations, done without special emphasis on small bowel technique, abnormalities were found in 11 examinations. This is 6% for positive findings, all of which were suspected clinically. However, in this same group 15 abnormalities were not found, these were subsequently turned up at autopsy or surgery, so 8% more were positive but were missed. Consequently, 14% of this series were positive for pathology. In our own series of 230 selected patients over a two-year period, 23% demonstrated pathology. These selected patients previously failed to provide an adequate explanation for persistent gastrointestinal symptoms on careful examination of the gallbladder, esophagus, stomach, duodenum, or colon. On the other hand, routine checks of these organs often provide unsuspected pathology. Why then do we not include the small bowel in these routine investigations?

As usual, there are several methods of examination of the small bowel, none of which are used routinely.

PREPARATION

We have found, along with others, that an empty colon facilitates the passage of the contrast through the ileum. Ileum—because here is the point of slowdown. Also an empty colon appears to allow the ileocecal valve to open more readily. An empty pipe open at both ends facilitates the speed of passage. Six tablets of Bisacodyl (Dulcolax®) with plenty of water, administered early the evening before the examination, seems to produce a good cleansing effect. Enemas and castor oil are not recommended.

CONTRAST MEDIA

The amount of barium is variable; however, it is generally agreed that a microscopic suspension of barium particles is better than plain barium sulfate. Incidentally, it is also more palatable. Several methods of administration have been advocated.

Golden:[2] 4 oz. barium plus 4 to 5 oz. water

Marshak:[3] 8 oz. barium plus 8 oz. water

Bendick:[4] 8 oz. barium plus 8 oz. soda water

Williams:[5] 8 oz. barium suspension in saline followed by 8 oz. of saline in one hour, the saline should be ice cold

Nice:[6] 10 oz. barium followed by 10 oz. of water after thirty minutes

Local: 8 oz. of barium for fluoroscopy of the stomach immediately followed by 8 oz. of barium which, in turn, is followed by 8 oz. of water at thirty minutes.

There are both advantages and disadvantages of these methods.

LARGE AMOUNT OF CONTRAST

A. Fills the stomach and promotes a continuous source of small bowel filling

B. Promotes a rapid filling and rapid flow through the small bowel

C. Water, saline soda water add a diluting and further stimulating factor to peristalsis

D. Causes overlap of ileal loops and difficulty in distinguishing separate loops or bowel walls

SMALL AMOUNT OF CONTRAST

A. Empties from the stomach more rapidly and allows better visualization of the ligament of Treitz area

B. Only a part of the bowel is filled at any one time so there is no overlapping of loops and the bowel wall is seen better

C. Provides less stimulation and therefore increases the time required

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OTHER CONSIDERATIONS

A. Saline should not be used with cardiac or hypertensive patients

B. Large quantities of fluid may develop edema in those patients on the verge of water imbalance

C. Water soluble contrast media has been advised by many as the contrast to use in bowel obstructions. Evidence has been compiled by reliable radiologists that they have never seen an inspissation of barium even in severe complete bowel obstruction.[7–12] Furthermore, the point of obstruction is outlined well by barium but is poorly seen, if at all, by a water soluble media. The fluid in the small bowel immediately proximal to the obstruction dilutes the barium and keeps it fluid. Consequently barium is advocated over water soluble media on all occasions.

TIMING AND METHODS

The patient should be placed in the right recumbent position immediately after fluoroscopy. Since recumbency in the department is difficult, the patient could be returned to his room and assume the right lateral position in his own bed. Outpatients would have to be accommodated in the department.

Method A.: Twenty minutes, after recumbency assumed, one 14 x 17 prone, one 14 x 17 supine of abdomen; fifty minutes, fluoroscopy of bowel loops; ninety minutes, spot films of terminal ileum, prone position

Method B: Routine thirty minute films of the abdomen in the prone position.

Method C: Thirty minutes after fluoroscopy, one prone and one supine film of the abdomen. Ninety minutes after fluoroscopy repeat as at thirty minutes. A compression device is used over the terminal ileum area.

BOWEL STIMULANTS

A controlled study[13] using a placebo and 0.5 mg. of neostigmine methylsulfate (Prostigmine®) subcutaneously, alternately, indicated only that the speed of passage of the barium mixture increased both methods indiscriminately. A physiologic 300 ml. test meal of barium mixed with homogenized corn oil passed through the small bowel with variable speeds. In the age groups 17 to 71 years the speed varied from fifteen minutes to two hours without any correlation with age.[14]

FACTORS

The penetration of the area should be as great as possible. Either 100 KvP with 300 Ma at one-tenth second or 150 KvP with 300 Ma at one-tenth second was used.

DEVICES

A. A Balsa wood block or an inflatable balloon or a plastic sponge placed in the area of the terminal ileum of the prone patient helps to dis-displace the loops of bowel so that overlap is less troublesome.

B. Small bowel enema[15] consists of intubation of the duodenum or jejunum followed by an injection of barium through the tube. This is a good idea but is time consuming and unpleasant to the patient.

C. Intubation with double lumen tube[2] consists of an inflated balloon which seals off the bowel and fills the proximal bowel with barium. Up to 600 ml. of barium may be used. Same objection as B.

D. Retrograde filling of the small bowel from the barium enema. This is difficult (try it).

ROENTGEN FINDINGS AND RESULTS

1. The single most overlooked lesion is the persistence of a single dilated loop of bowel proximal to the lesion.[7–12,16]

2. Another factor not utilized is the supine film. This is particularly helpful in diagnosis of abnormalities in the base of the small bowel mesentery.

3. Flocculation is regarded by many radiologists as the best sign of small bowel disease. Rarely is flocculation present in the normal patient if a large volume of a microscopic particle suspension of barium is used.

4. Loss of jejunal folds is a loss of the feathery pattern.

5. Puddling, clumps of barium of 2 to 4 mm. diameter left after the bolus of barium has passed

6. Segmentation is clumping of barium in the loops not in continuity with the barium in the adjacent loops

7. Dilatation of small bowel loops,[7–12,16] if over 3 cm. in diameter in the jejunum and over 2.5 cm. in the ileum

CONCLUSION

Using a composite of the aforementioned ideas, a possible method to more completely examine the small bowel along with the routine stomach examination is suggested.

Empty the gastrointestinal tract, use microparticle barium in large amounts, and follow the routine filming of the stomach with prone and supine abdominal films.

(References continued on page 246)

Vesicoureteral Reflux: A Brief Review

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EACH GENERATION of urologists seems to find a favorite subject to debate, although not as acrimoniously as in the not too distant past, when personal insults were often exchanged. Fields for argument have included questions of priority in the development of instruments and operations, the catheter versus prostatectomy, the merits of one type of prostatectomy compared to others, the place of transurethral resection, and so forth.

Presently, the treatment of vesicoureteral reflux is evoking sharp differences of opinion, although the phenomenon of reflux has been well known for a long time. Galen (150 A.D.) is said to have observed it in a living dog. Its nature and causes were reviewed by Lewin and Goldschmidt in 1893 and by Warschauer in 1901. Sampson described the ureterovesical valve in 1903. Kretschmer in 1916 satisfied himself that reflux was an abnormal phenomenon in children. Graves and Davidoff reported extensive experimental investigations in 1923, and Bumpus studied its occurrence in 1,060 cystograms in 1924.

Current intensified interest in it began around 1952 with Hutch's presentation of what was apparently the first serious attempt to correct reflux surgically. This interest has been increased by widespread recognition of the fact that vesicoureteral reflux requires consideration as an etiologic or complicating factor in every urinary infection, particularly in young girls.

Factors tending to prevent reflux are well known. The peristalsis in a normal ureter is important. Figure 1 is an excretory urogram made fourteen years after a Boari operation, which removes the valvular mechanism at the entrance of the ureter into the bladder, yet the patient's ureter and kidney have remained normal.

Figure 2 is a diagram of this valvular mechanism: the obliquity of the intramural ureter and the flap composed of ureteral and vesical mucosa which surmounts the ureteral orifice (Sampson's ureterovesical valve). A rise in intravesical pressure compresses them. A third factor is the intravesical pressure which, between urinations, is only a few centimeters of water—very little more than that which prevails in the renal pelvis and ureter. During normal micturition the intravesical pressure may rise as high as 40 cm. of water; it is obvious that its transmission to the kidney will have adverse effects, both by raising pressures and by carrying infected material from bladder to kidney.

Certain situations are known to favor the development of reflux; others are known to aggravate its effects. Some females are born with ureters which run straight through the vesical wall and hence, have no valves at all. Other disorders thin the wall of the bladder and so shorten and straighten the intramural ureter; the ex-



Fig. 1. Urogram fourteen years after Boari operation

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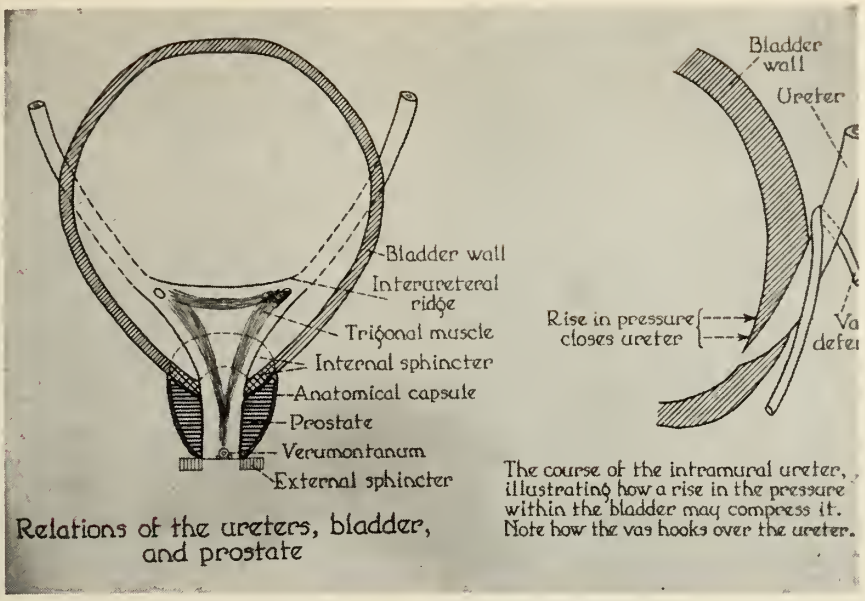


Fig. 2. Ureterovesical valve and oblique intramural ureter

cessive dilatation of the ureter of congenital ureterectasis may cause its intramural portion to gape; some patients are born with thin bladders (megacystis syndrome); the consequences of meningomyelocele and other disorders of the spinal cord, as well as of neglected obstruction at the vesical outlet (valves, strictures) also thin the detrusor.

Infections, both acute and chronic, may make the intramural ureter rigid and permit reflux. Ureteral meatotomy and unsuccessful reimplantation of the ureter may have similar effects.



Fig. 3. Dilatation of terminal ureters due to vesicoureteral reflux



Fig. 4. Reflux on right in urogram appearing with full bladder after upper tract had emptied

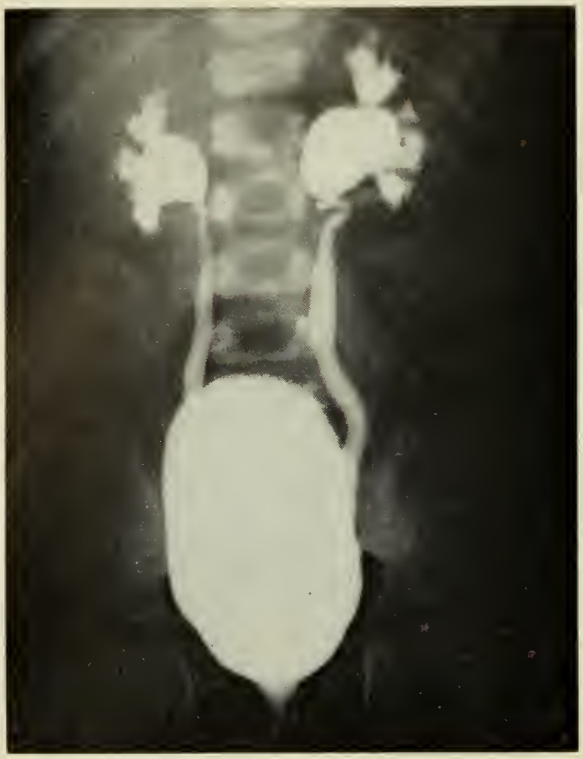


Fig. 5. Reflux in a voiding cystourethrogram

The sequelae of a preexisting regurgitation are obviously aggravated by the elevated voiding pressures which may accompany obstruction at the vesical outlet.

There is no symptom complex which is characteristic of vesicoureteral reflux. It is usually discovered during investigation of chronic or recurrent acute urinary infections, mainly in little girls. An occasional adult will complain of renal pain during or just after micturition.

It is my earnest belief that painstaking diagnostic measures should be instituted after a patient's first urinary infection, unless it unquestionably resulted from urethral instrumentation. One should also consider employing these measures in unexplained renal insufficiency.

The excretory urogram is often suggestive but rarely diagnostic. The earliest sign (Fig. 3) is dilatation, perhaps transitory, of one or both terminal ureters, but this may also occur with other disorders. Later, of course, both ureter and renal pelvis may exhibit more or less advanced dilatation. One can sometimes demonstrate reflux by altering the technique of the urogram: films are exposed at intervals until the kidneys and ureters are nearly empty. One next waits until the patient has a strong desire to void, then



Fig. 6. Vesicoureteral reflux with dilatation of lower segment of duplicated pelvis (urogram)



Fig. 7. Urogram of patient in Figure 6 after three years of medical therapy, patient asymptomatic

exposes a last film, preferably during urination. Refilling of one or both ureters is diagnostic (Fig. 4).

One usually depends upon the voiding cystourethrogram (Fig. 5), either still or cinefluorographic. The principal advantages of the latter are that the urinary tract is observed over a longer period than with stills and that, if properly done, the cine permits observation of ureteral peristalsis, as well as of the behavior of the bladder and urethra. Fleeting, incomplete reflux observed during fluorography requires that its importance be proved by a period of observation.

Cystoscopy is useful to confirm or disprove impressions gleaned from the preceding studies; it involves a search for urethral obstruction, valves, trabeculation, and "straight through" intramural ureters. In the last analysis, one has to put everything together; a period of trial, error, and observation may be necessary. Snap decisions should be avoided.

There is no unanimity of opinion about choice of treatment. It is generally agreed that obstruction to urination must be identified and eradicated (urethral dilatation, internal urethrotomy, plastic widening of the internal meatus), but beyond that opinions diverge. One group defends medical treatment and decries surgery. Another defends operation upon the vesical outlet and condemns ureteral reimplantation. Still another favors reimplantation of the refluxing ureter, while a last group insists upon reimplantation of both ureters and revision of the vesical outlet even for mild reflux into one ureter. One suspects that the truth is to be found somewhere in the middle, but these questions have not really been entirely settled except in the minds of a few.

Medical treatment consists of long-term antibacterial therapy plus timed voiding: the patient urinates every three hours during the day and once in the middle of the night. Stephenson has advocated double and triple voiding as well: the patient urinates, waits a minute or two, and then tries again. This maneuver is based on the assumption that the urine which regurgitates to the kidneys during micturition will descend to the bladder shortly thereafter. Manifestly, such regimens are not available in the untrained infant, or in the patient with a nonfunctioning bladder.

These measures may control many mild cases (Figs. 6 and 7), but careful follow-up is essential, so that tactics can be changed if lasting cure does not result. If conservative treatment re-



Fig. 8. Degree of reflux indicating primary reimplantation



Fig. 9. More advanced renal damage. Question of nephrostomy came up, but primary reimplantation succeeded.

lieves symptoms, it becomes progressively more difficult to implement as the asymptomatic interval lengthens. Moreover, it will fail unless the mother is intelligent, cooperative, and persistent.

When does one direct attention to altering the vesical outlet (internal meatus, prostatic, and female urethra)? Removal of valves of the posterior urethra is ordinarily the first step in the treatment of that disorder. Strictures of the meatus and urethra, as well as contraction rings (distal urethral stenosis) in the female urethra must be corrected in order to reduce voiding pressures. However, if an open operation such as Y-V plasty is required for contracture or rigidity of the internal meatus, I believe that ureteral reimplantation should be done at the same time. I am not yet entirely convinced, as some are, that both ureters should be reimplanted for unilateral reflux. My attitude is based upon the fact that one cannot count absolutely upon a perfect or near-perfect result from any type of reimplantation.

I do believe, however, that if reimplantation is done, the vesical outlet should ordinarily be widened at the same time, since it is so difficult to be absolutely certain that no impediment to urination exists.

Vesicoureteroplasty is definitely indicated when conservative measures have failed or are unavailable, in the presence of total reflux, when ureteral and pelvic dilatation are mild to moderate, and renal function is but little impaired. Prospects of success are excellent (Fig. 8).

There are cases (Fig. 9) in which success is less certain. Here, with considerable dilatation and definite impairment of renal function, the question of preliminary nephrostomy comes up. Since nephrostomies are at best a nuisance and at worst a menace (infection, stones), one is inclined in case of doubt, to employ primary reimplantation after making it clear to the parents that temporary nephrostomies may become necessary in the immediate postoperative period, because of edema about the anastomoses. However, primary reimplantation without nephrostomy did succeed in the patient illustrated in Figure 9.

Other patients have so much dilatation with impairment of peristalsis and of renal function that primary reimplantation would clearly be foolhardy (Fig. 10). Here preliminary nephrostomies are done. Close follow-up is essential and includes, in addition to periodic assessments of renal function, measurement of transit time between renal pelvis and bladder and, finally, cinefluoroscopy to assess ureteral peristalsis. The period of nephrostomy drainage may be as long as a year.



Fig. 10. Degree of damage clearly requires nephrostomy.



Fig. 11. "Cuff" ureterostomy

If renal function improves and active ureteral peristalsis can be demonstrated, one may narrow the ureters by excision of longitudinal strips followed by suture around catheters (taking care not to narrow them too much); they are then reimplanted submucosally, but nephrostomy is maintained until it is certain that the revised ureters function adequately; this, too, may take some time. It is in this group that the proportion of failures is highest; however, since the alternative is some form of urinary diversion, it is worth taking a chance in case of doubt.

In some of the patients with preliminary nephrostomy, neither peristalsis nor renal function improve. Here one resorts to some form of urinary diversion which does not require inlying catheters of any kind, since their protracted presence predisposes to infection and lithiasis. If the ureters are not "paper thin," one may employ the form of cuff ureterostomy in which the ureters are anastomosed to one another as they course through the abdominal wall (Figs. 11 and 12) in order to make the stoma large and so to obviate the use of catheters. If the ureters are very thin walled, pyeloileostomy is the only useful alternative.

In an occasional patient, usually because of meningomyelocele, the bladder cannot be made to function. Here one resorts to one of the diversionary procedures just mentioned without considering either nephrostomy or reimplantation.

The first vesicoureteroplasty to offer any prospect of correcting reflux was apparently that of Hutch (1952, Fig. 13). While it often succeeded, it was soon discovered that too frequently a constricting scar formed beneath the regenerated mucosa where the ureter entered the vesical lumen. It seems likely that this relates to the fact that the Hutch operation leaves a segment of bare ureter within the vesical lumen, to be covered by proliferation of the mucosa of the bladder. Perhaps this evokes more scar than does a procedure which buries the intramural ureter, so that mucosal regeneration is unnecessary.

Hutch's report has been followed by a rash of new operations—many intended to improve results; others obviously aimed at getting the authors' names into the literature.

The most successful reimplantation thus far has been that of Politano and Leadbetter (Fig. 14). However, when the caliber of a much dilated ureter has to be diminished, I believe that this should be done extravesically so that kinks can be straightened and the narrowing carried well up toward the renal pelvis. One should

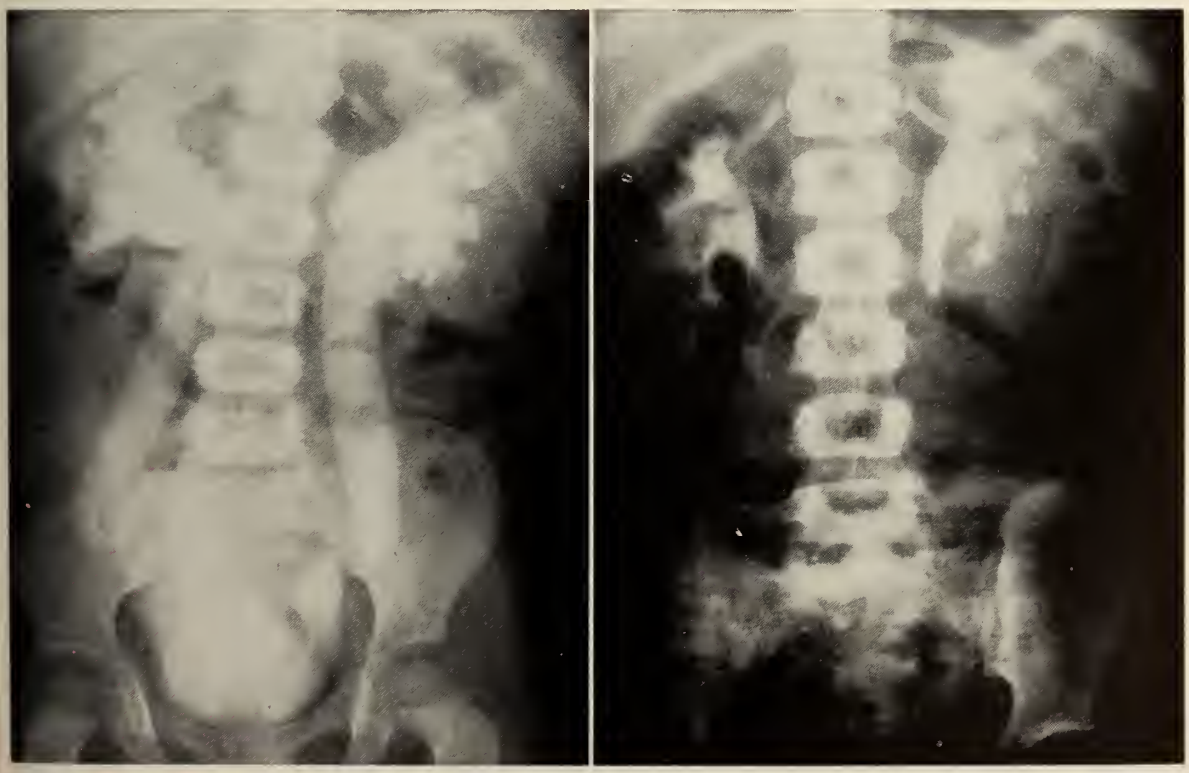


Fig. 12. Pyelograms before and forty-two months after ureterostomy. Same patient as in Figure 11

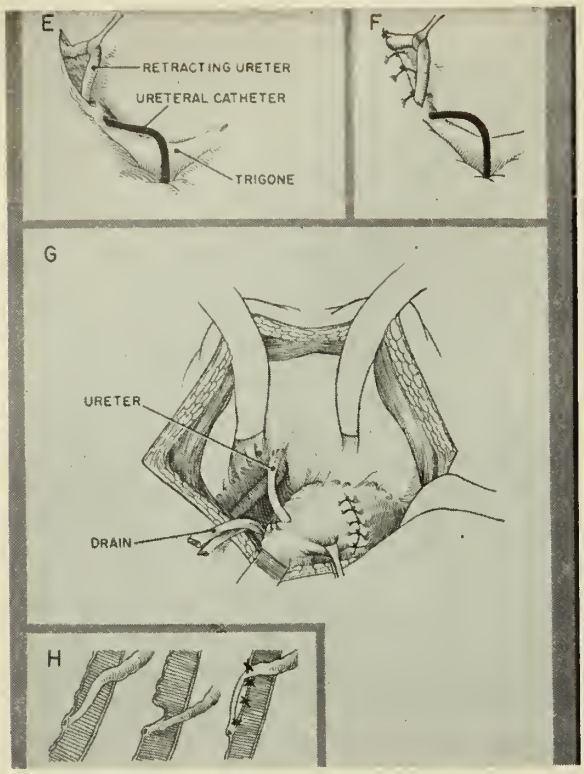


Fig. 13. Hutch's operation. Reproduced with the permission of Flocks, R.H., and Culp, D.: Surgical Urology. Chicago: Year Book Publishers, 1961, p. 149.

restrain the impulse to excise too much of the ureter, bearing in mind that it was dilated by the pressure of the reflux. When this factor is removed, the ureter may shrink to some extent; if surgical narrowing is overdone, it will become too small and the operation will fail.

Only 2 other operations out of the many devised will be mentioned, even though more experience with them is needed, and others have merits of their own. Bischof's method (Fig. 15) has the advantage that, if it fails, it will probably do no harm. The same seems to be true of ureteral advancement, of which Williams' is typical (Fig. 16). However, neither can be done if, as is often the case, the ureteral orifices are close to the internal urinary meatus.

The objective of all of these operations is to leave a segment of supple ureter lying beneath normal mucosa and upon solid detrusor muscle, so that a rise in intravesical pressure will compress it. Success, therefore, depends not only upon good technique, but also upon "kind healing" so that a small but unavoidable element of luck is involved.

Potential complications include: injury to intraperitoneal structures during dissection; displacement, angulation, and extrinsic compression of the ureter; stricture from devascularization,

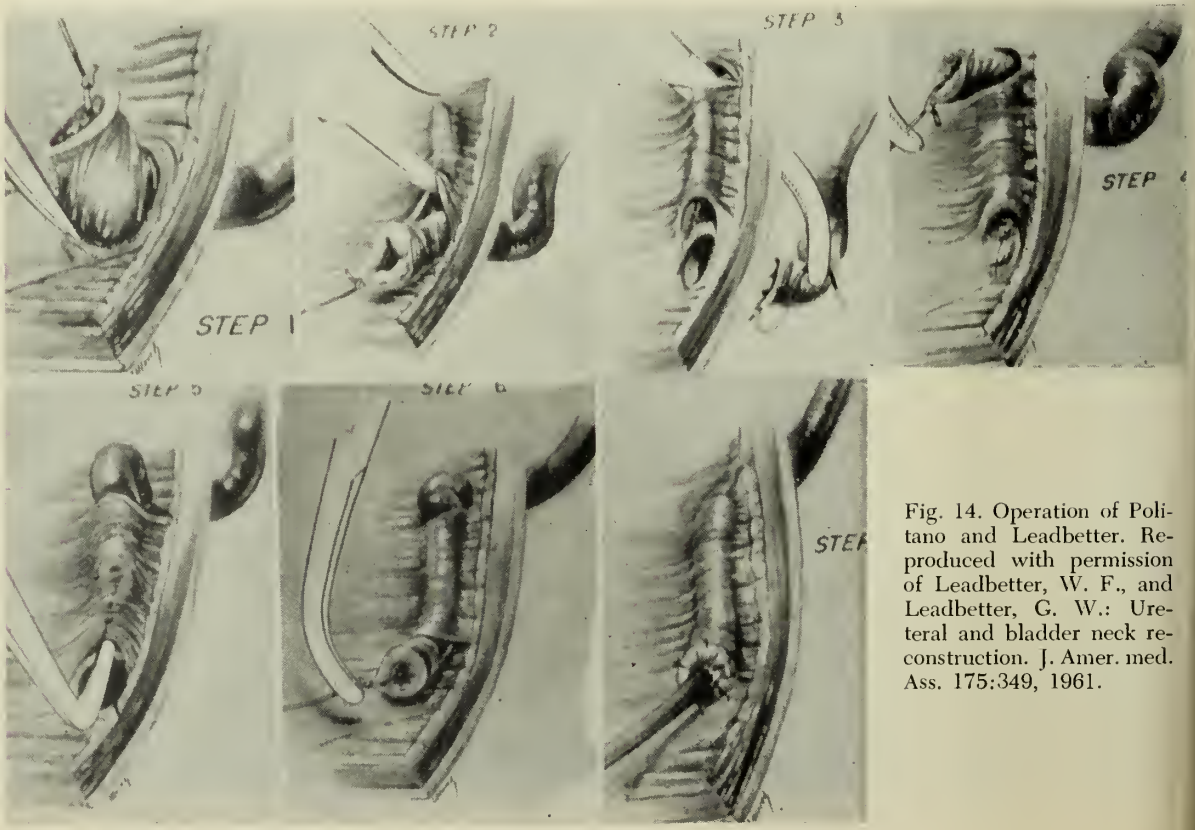


Fig. 14. Operation of Politano and Leadbetter. Reproduced with permission of Leadbetter, W. F., and Leadbetter, G. W.: Ureteral and bladder neck reconstruction. J. Amer. med. Ass. 175:349, 1961.

infection, or overzealous reduction of the ureteral lumen; and a tunnel which is too long (obstruction) or too short (reflux continues).

Danger always exists that, following reimplantation, the local reaction will be severe enough to occlude the ureteral lumen and necessitate temporary nephrostomy, a possibility which should be explained beforehand.

Herewith is a brief summary of our experience with the operation of Politano and Leadbetter for vesicoureteral reflux at the University Hospital during the past twenty-two months; I doubt that any follow-up shorter than five years justifies use of the term "cure." It is of necessity scarcely more than a preliminary report. Let me emphasize that our material includes rather a high proportion of unfavorable cases in which reimplantation was done despite a real possibility that urinary diversion might be required; these cases represent an effort to avoid committing the children to life-long use of collecting devices. However, an occasional complication as well as failure will occur (as is the case with any surgical operation of any magnitude) even in apparently favorable circumstances. There are surgeons who "never have any complications"—but what about their patients?

The material is limited to those who had vesicoureteroplasty for reflux. There were 39 females and 15 males. The youngest patient was 1 month old, the oldest was 44 years of age. The average age was 11½ years. Thirty-nine of the 54 patients had plastic widening of the vesical outlet done at the time of reimplantation. The operations were performed by 6 different surgeons (3 members of the staff and 3 residents).

The status of the upper urinary tract as judged by preoperative excretory urography was normal in 10 (8 females, 2 males); slightly dilated in 23 (19 females, 4 males); and there was advanced pelvic and ureteral dilatation in 23 (12 females, 11 males).

Five patients (4 males, 1 female) required preliminary nephrostomy because of the severity of pelviureteral dilatation.

Ten suffered significant surgical complications during immediate convalescence. Seven (4 males, 3 females) required temporary postoperative nephrostomy because of inflammatory stenosis of the reimplanted ureter. In 1 female infant there was postoperative intestinal obstruction requiring reoperation, after which the bladder broke open.

There were 5 patients (1 female, 4 males) who required secondary reimplantation of 1 ureter because of cicatricial stenosis; these were successful.

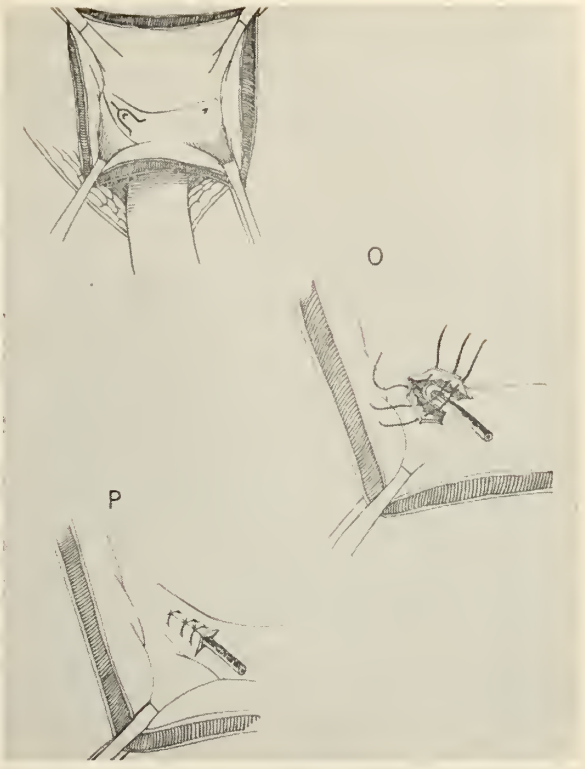


Fig. 15. Bischof operation. Reproduced with permission of Flocks, R. H., and Culp, D.: Surgical Urology. Chicago: Year Book Publishers, 1961, p. 153.

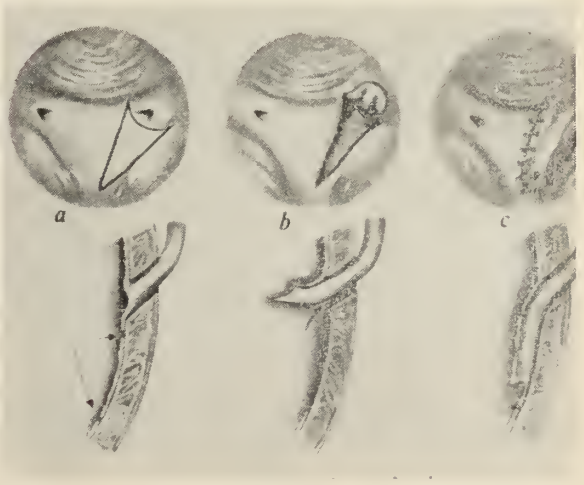


Fig. 16. Ureteral advancement of Wilhams

There were 5 frank failures (2 females, 3 males). Two of these required nephrectomy, although the reimplantation was successful on the opposite side. Two others (both males) required permanent urinary diversion (1 ureter-

ostomy, 1 ileac diversion). The fifth, a woman of 44, had rather severe chronic bilateral pyelonephritis and left renal calculi, both attributed (perhaps falsely) to vesicoureteral reflux. Some months after passing 1 stone and having pelvionephrolithotomy for the other, bilateral vesicoureteroplasty was done. Six months later, despite absence of complaints, the urea nitrogen had risen from 18 to 35, although both ureters were easily catheterized. This represents an error in judgment—the reflux should have been treated conservatively.

One patient had persistent reflux at the six month follow-up, but without complaints or deterioration in the urogram.

Two patients, after remaining well without medication for eight months, developed acute cystitis requiring resumption of chemotherapy, and several others have positive urine cultures without pyuria or complaints.

It appears that, aside from errors in technique or judgment, factors most likely to defeat attempts at ureteral reimplantation for reflux are: advanced pelviureteral dilatation, neurogenic vesical dysfunction, and chronic cicatricial pyelonephritis.

SUMMARY AND CONCLUSIONS

1. During the past twenty-two months, 56 patients have undergone ureteral reimplantation for reflux by the method of Politano and Leadbetter.

2. There were 47 tentative successes, although 5 of these required secondary reimplantation of 1 ureter before success was achieved.

3. Failure was due chiefly, but not entirely, to advanced disease in the upper urinary tract before surgery.

4. Continuous follow-up will be required for a much longer period before one can say that neither relapse nor complications can occur.

Continued from page 237

WHAT ABOUT THE SMALL BOWEL?

Milton R. Gilchrist, M.D.

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Chondritis as a Postoperative Complication

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Wound infections of most kinds are an annoying blemish to convalescence requiring a number of additional dressings, which embarrass the surgeon and irritate the patient. Sometimes they go beyond this and become a great trouble, chronic beyond all expectation, demoralizing to the patient and surgeon alike. There are well-known causes of wound infections with this troublesome chronicity, for example: deep silk suture abscesses, fistulas from visceral organs, fungus and tuberculosis infections, osteomyelitis, empyema, and bronchopleural fistulas. Perhaps less well known as a cause are infections of the costal cartilages. I have encountered four patients with this complication, and they form the basis of this report.

CASE REPORTS

Case 1. A 47-year-old man with bilateral bullous emphysema underwent resection of a number of large bullae in the right lung with uneventful recovery. Six months later he underwent a similar operation on the left lung, but there was a prolonged air leak after this procedure. High negative intrapleural suction was used; the operation had been done before our work suggesting the superiority of low suction pressure in such cases.[1] Also, the intrapleural catheter was in a position too high in the chest, and after nineteen days of air leak, the catheter eroded the right brachial artery. At emergency thoracotomy, there was some local infection at the site of the air leak. He developed a late wound infection.

Three months postoperatively, chondritis was recognized and the seventh through tenth cartilages were removed on the left (Fig. 1). Three months later infection was still present so the sternum was curetted at the medial end of the incision. The infection persisted. Finally, one year after the cartilages on the left had been resected, infection had clearly spread to the right and two cartilages on the right were removed. Following this, the wound healed slowly.

Case 2. This 66-year-old man had an epiphrenic diverticulum of the esophagus which caused a long history of dysphasia. The diverticulum was excised but the repair leaked three days postoperatively. He underwent a thoracotomy for repair of this leak and developed a wound infection. Six months postoperatively chondritis was recognized as a cause of the persistent infection and two cartilages on the left were excised (Fig. 2). Two months following this operation, it was necessary to excise three more cartilages and, finally, after another delay of two months, the left fifth cartilage was removed. The wound then healed.

Case 3. This 34-year-old woman underwent a number of operations for a biliary stricture which had developed following a cholecystectomy at another hospital. A hepatojejunostomy was performed. Five months later it was necessary to do another operation for the recurrent stricture, and eleven months following this a reoperation was done with insertion of a T tube. Infection of her midline abdominal wound was apparent two months later and an infected xiphoid cartilage was resected. Bilateral lower chest pain persisted, however, and the wound continued to drain. The difficulty was then recognized as being a bilateral chondritis. Therefore, the right conjoined cartilages were excised three months after resection of the xiphoid, and one month following this the total left conjoined cartilage was removed (Fig. 3). Two months later pinch grafts were placed in the wound and it finally healed satisfactorily, though there is considerable flexibility of the anterior chest wall.

Case 4. A 35-year-old man had a mediastinal thyroid removed through a sternum-splitting incision. Two weeks following this operation while undergoing a bronchoscopy for recurrent atelectasis, the sternal incision split open. A wound infection developed which was debrided but failed to heal. We did not consider chondritis at first because the cartilages had not been exposed, but fifteen months after the original operation, chondritis was finally identified and the fifth, sixth, seventh, and eighth costal cartilages were excised on the right (Fig. 4). Two weeks following this, a reoperation was done merely to reopen the wound. Two months later, infection was identified on the left and the single fourth cartilage was excised. Following this the wound healed.

REVIEW OF THE LITERATURE

Others who have reported their encounters with this disease have had difficulties similar to those which we have described. The condition is chronic but curable, in most cases, when the involved cartilages have been completely removed. A list of the major reports on this condition since Murphy's report in 1916[2] is given in the table along with the number of documented cases reported by each. Perhaps one of the best articles on the subject was written by Moschcowitz in 1918.[3] He recognized the essential problems of the disease and pointed out that

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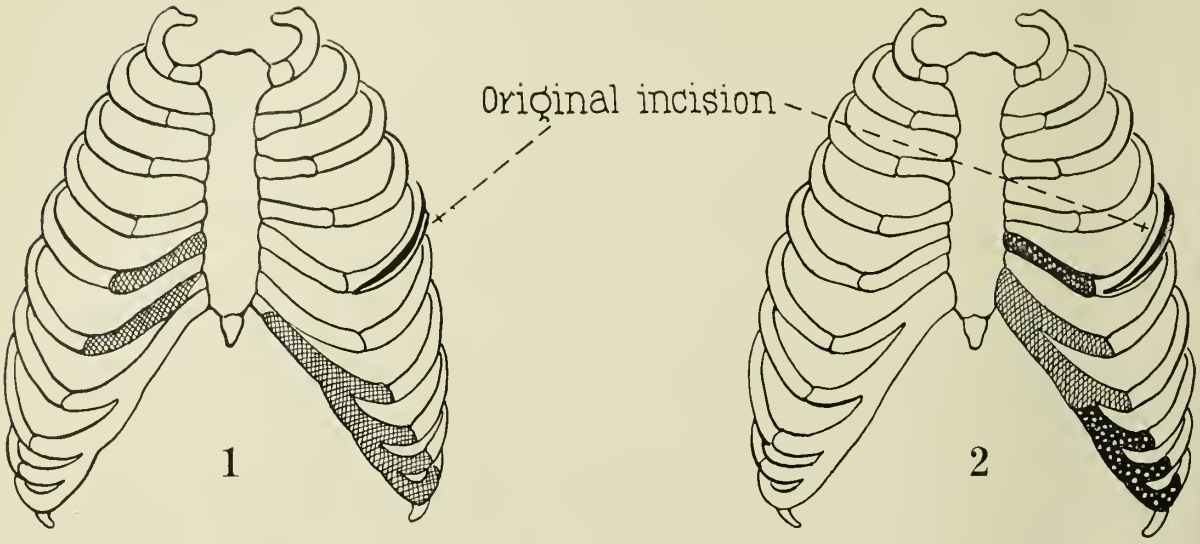


Fig. 1. Diagram illustrating the cartilage resections necessary in Case 1. The original wound is indicated by the line over the left fifth rib.

Fig. 2. Case 2. The sixth and seventh cartilages on the left were excised. Two months later, the eighth, ninth, and tenth cartilages were excised. Finally, the fifth cartilage was removed.

chronicity was not due so much to the type of infecting organism as to the fact that cartilage, an avascular tissue, was infected. He also pointed out that complete removal of the involved cartilage was required.

The disease is rather uncommon with perhaps a slightly increased incidence, or at least a slight increase in the incidence of reporting, in recent years. The sex incidence in the reported cases is 4:1, males to females, similar to the 3:1 ratio in our four cases. The age incidence is entirely adult in the reported cases as well as in ours.

ETIOLOGY

Before 1940, most of the cases were spontaneous in origin and due to tuberculosis, though some were due to typhoid or paratyphoid. None of the cases reported before this time represented complications of surgery. In contrast, since 1940 about one-half of the reported cases have been surgical complications and this ratio appears to have increased in recent years. All of our cases were complications of surgery.

ANATOMY

The extent of the fusion of the costal cartilages of ribs 5 through 8 or 9 is a critical anatomical feature of this disease for it dictates the extent of the resection which is required. Isolated cartilages, such as those of ribs 1 through 4, may be excised individually if infected. In contrast, if any one of the conjoined lower cartilages is infected, all require resection. Also of anatomical importance is the structure of the xiphoid. If it is partly cartilaginous, it may promote a bilateral spread of the condition. Hyaline cartilage is avascular and thus tends to behave as a foreign body when involved in an infection.

PATHOLOGY

The disease may be classified into three gross pathological types of increasing severity. First is involvement of a single cartilage, usually one of the first four cartilages on either side. The second type is involvement of the conjoined cartilage on either side, while the third type would include cases of bilateral involvement. Three of our four cases fall into the third classification. When

CHONDRITIS

Author	*Date*	*No. of cases*
Murphy[2]	1916	2
Moschcowitz[3]	1918	5
Brock[5]	1935	7
Chaklin[6]	1937	14
Bisgard[7]	1939	1
Siler[8]	1942	1
Maier[9]	1947	4
Royster and associates[10]	1948	1
Koontz[11]	1949	1
Shutkin and Cary[12]	1951	1
Brown and Trenton[13]	1952	7
Pontius and associates[4]	1959	17
Wilcox[14]	1965	3

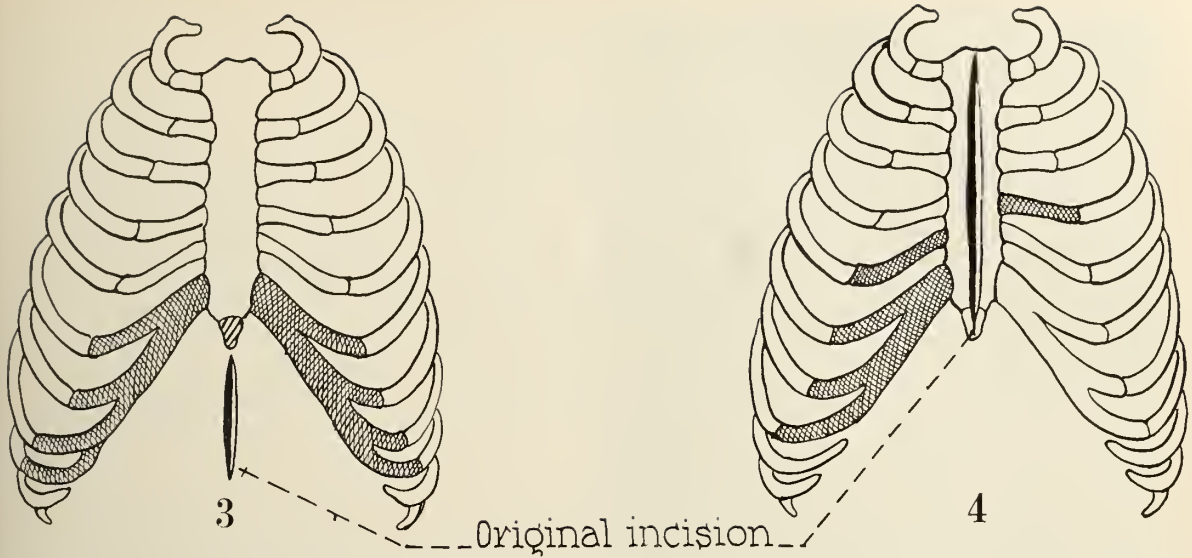


Fig. 3. Case 3. The midline abdominal wound, which was the source of the infection, is illustrated. The sixth through ninth cartilages on the right were excised, and the same operation was done on the left as a second stage.

Fig. 4. Case 4. Original infected incision was a sternum-spliting incision. Cartilages were excised on the right. The left fourth cartilage was removed two months later.

exposed, the diseased cartilage is frec of its perichondrium and bare, and it has an irregular, rat-eaten appearance suggesting that some of its substance has been dissolved or destroyed by the infectious process. If this dissolution does occur it occurs so slowly, however, as to be of no practical value in resolving the process. There is no record of an entire costal cartilage being absorbed by the infection. The cartilage does not disappear, neither does it form sequestra to be extruded from the wound as in osteomyelitis.

BACTERIOLOGY

The following organisms have been associated with chondritis: tubercle bacillus, typhoid, paratyphoid, actinomycosis, streptococci, staphylococci, Escherichia coli, pneumococci, Nocardia, and pscudomonas. Recently, P. aeruginosa may be found more frequently in these as in other wound infections as other organisms succumb to antibiotics. Three of the seven cases in thc Mayo Clinic series[4] in which bacteriology was described had a pseudomonas infection, as did all of our four cases. Pseudomonas is described as a common saprophyte not infrequently found as an opportunistic or secondary invader in wounds or ulcers that have not healed promptly. Surgeons have encountered it with increased frequency in the antibiotic era.

CLINICAL PICTURE AND DIAGNOSIS

Clinically the disease manifests itself as a draining sinus in the region of the costal cartilages usually associated with considerable local pain and tenderness. There may be some general debility, but this has not been greater than that associated with other wound infections in my experience. The differential diagnosis is apt to be simply that of a wound infection without associated chondritis or one with chondritis. The surgeon, usually of an optimistic frame of mind by nature, is apt to assume that he has an ordinary wound infection to treat until inordinate chronicity or previous experience finally brings him to accept the diagnosis of chondritis.

Roentgen examination helps rule out the diagnosis of empyema but usually provides no specific diagnostic findings. A sinogram will show only a nondescript sinus, though occasionally it may outline the location of the diseased cartilage and thus be diagnostic, as it was in our fourth case (Fig. 5).

TREATMENT

Treatment is operative and basically the same as advocated by Murphy in 1916[2] and Moschcowitz in 1918.[3] The entire diseased cartilage should be removed. When cartilages of adjacent ribs are continuous and fused, as is normal in the lower ribs, they, too, must be excised so that no bare cartilage remains in the infected wound. Thus, the entire conjoined cartilage must be excised if any of the fifth through eighth, ninth, or tenth cartilages is diseased. Exceptions to this have been made successfully if the fusion between the cartilages is small and surrounded by



Fig. 5. Case 4. Iodized oil (Lipiodol®) was injected in the draining sinus and it promptly dispersed along the beds of two of the infected cartilages.

healthy tissue, but the risk of persistent chondritis is great and it may require further operations, as in our second case. Though some authors have advocated closure of the wound after excision of the diseased cartilage, we have packed the wound open and cannot state from our own experience when primary closure is indicated.

Apparently the cartilages have not regenerated in our patients for the lower chest wall remains unusually flexible. This is somewhat annoying to the patient but not disabling. All of our patients have been seen at least six months after complete healing of the area and there has been no late breakdown.

The total duration of the illness has been distressingly long, ranging from twelve to eighteen months in our four cases. One private patient required thirty-nine days of hospitalization, and the other three patients spent from one hundred fifty-nine to one hundred seventy days in a Veterans hospital recovering from the disease.

To prevent this complication the surgeon obviously should avoid producing infected wounds in the general region of the costal cartilages. Infections should not be drained through the area of the cartilages, and chest catheters should not be brought out through stab wounds near the cartilages. If an infection does develop, the best result is achieved through prompt recognition and complete resection of the diseased cartilages.

SUMMARY

Four cases of costal chrondritis developing as complications following chest or abdominal surgery have been described. Through aggressive removal of the diseased cartilages, healing was finally obtained. The disease lasted from twelve to eighteen months before complete healing.

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The Objective Evaluation of Auditory Function

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An objective method of determining auditory acuity would have great value to the clinician. Previous investigations in this area have focused principally on methods of recording electroencephalographic or cortical responses to auditory stimuli. In order to separate the auditory responses from the myriad of other random electrical activity, it has been necessary to apply computer techniques.

At Mayo Clinic, Cody[1] has correlated auditory evoked cortical thresholds with audiometric thresholds in a number of patients with normal and impaired hearing. The technique involves attaching tinplate electrodes to the skin with collodion. The active electrode is placed at the vertex, with the reference electrode near the inferior pole of the auricle of the ear being tested. The ground is usually placed on the forehead. Pure tones generated by a standard audiometer are then delivered to the ear through the usual earphones. Cortical responses are passed by way of the electrodes to appropriate amplifiers and then into the computer of average transients (the Mnemotron CAT 400B). Random electrical activity is erased by the computer compilation of multiple tone responses. Usually from 50 to 100 passes are used. The total period of time spanned by a typical tracing, as analyzed by the computer, is about 500 msec. The first peak appears around 75 msec. and the second, around 300 msec. The size of these peaks diminishes as cortical threshold is approached.

At the present time, Cody notes a fairly good correlation between cortical threshold and normal audiometric threshold. This method of threshold determination should be useful in infants, young children, and malingerers. In testing children, Cody has noted some changes in peak latency and shape when sedation is used, and this finding has been confirmed in adults during sleep. The cortical method, as outlined, can indicate whether or not the patient hears in a given ear and if so, at approximately what level.

Objective methods for evaluating hearing do not allow precise delineation of pathology to a specific focus in the sensorineural system. Sensorineural hearing loss has been a total diagnostic wastebasket for so long that it has almost become accepted as a single diagnostic entity. In fact, it may arise from faulty function, individually or collectively, in the cochlear structures, the nerve pathway, or the brain.

At St. Joseph's Research Laboratory we have been working toward a more complete and specific diagnostic sequence for objective evaluation of hearing. We propose analysis of cochlear function obtained through recordings of cochlear microphonics from skin or tympanic membrane electrodes. The instrumentation required for this type of recording is similar to that outlined by Cody but includes increased amplification and use of filters.[3] Our basic work has been done on cats and monkeys.

The cochlear microphonic, as defined by Davis,[2] is an alternating current response of the hair cells to acoustical stimulation. It is believed that the specific source is the external hair cell.

Microphonics have been recorded in human beings during middle-ear surgical procedures[4] using a similar method. However, a nonsurgical method is necessary if it is to be used clinically. For this reason, we have developed the technique in experimental animals which uses only skin or tympanic membrane electrodes or both.

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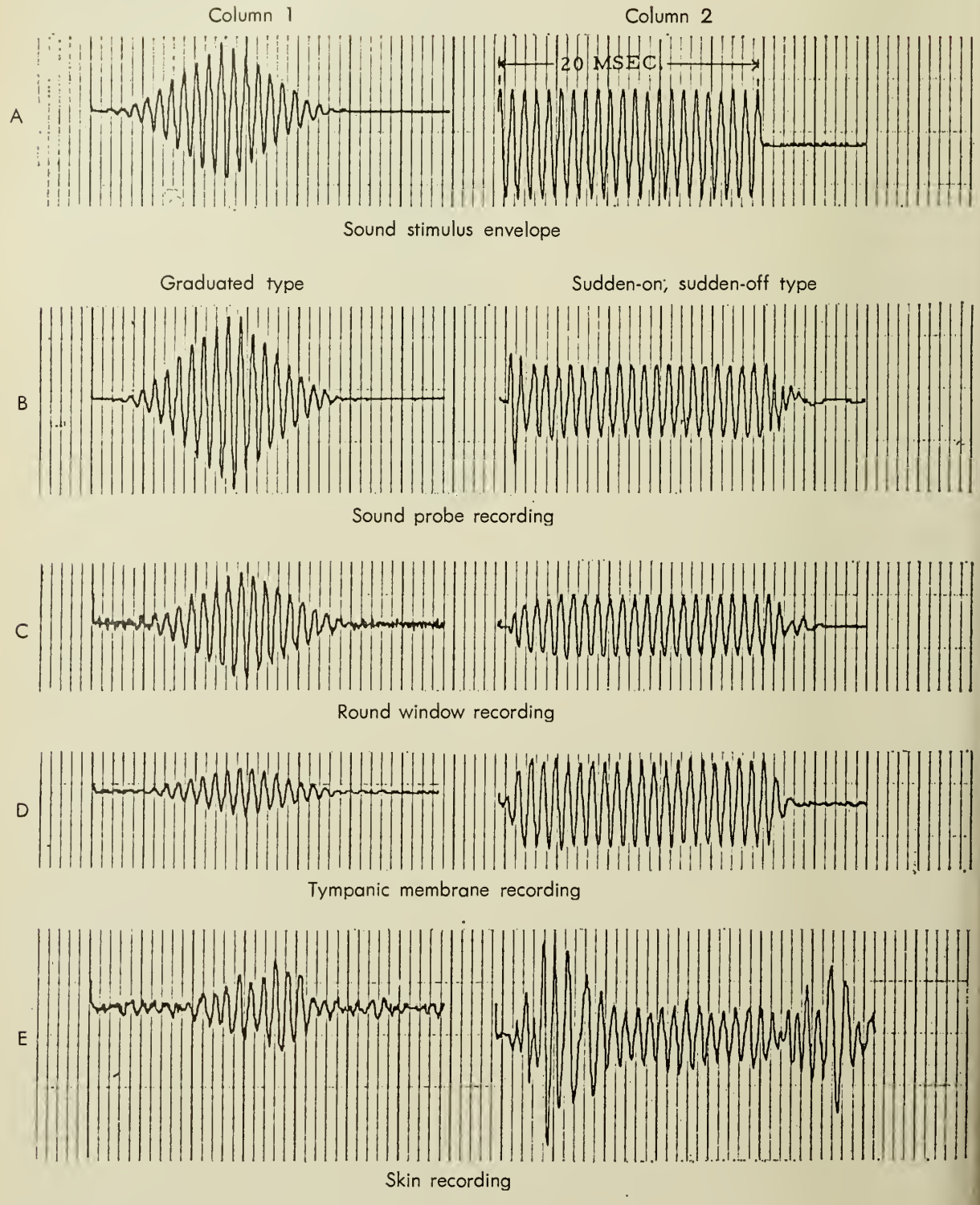


Fig. 1. Typical recordings of cochlear microphonics with two different sound stimulus envelopes

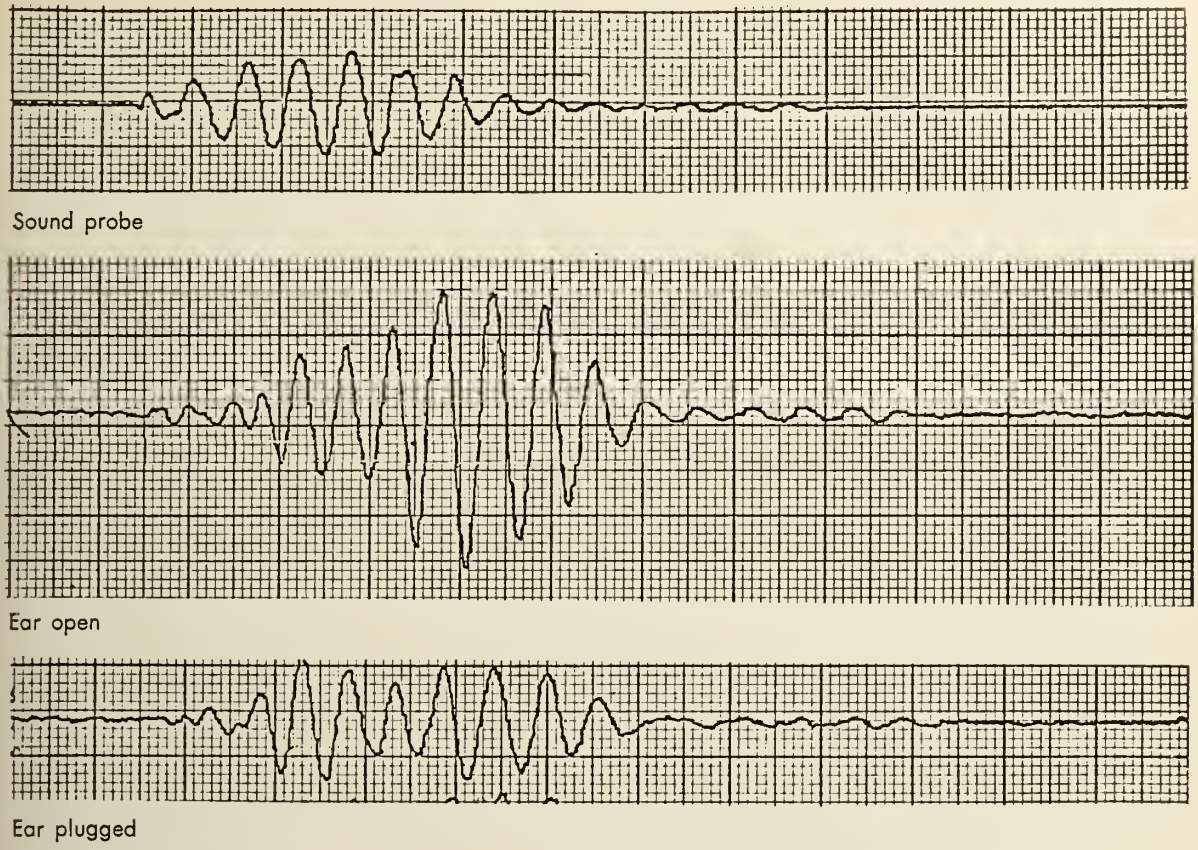


Fig. 2. Cochlear microphonic recorded from skin electrodes showing reduction of amplitude when the external ear of a cat is obstructed with a cotton and vaseline plug

Such recordings require the use of high-gain instrumentation (amplification up to 1 million).[3]

Precise electrode placement for obtaining microphonics from the cat's skin is not critical. An active electrode is placed in the inferior postauricular area, a reference electrode in the superior preauricular area, and a ground on the back of the neck. The electrodes are simple steel hypodermic needles which are inserted beneath the skin. For tympanic membrane recordings, the ear canals are prepared with a saline wash and a small amount of electrocardiographic paste is placed in the anterior sulcus. A ball-tipped, shielded platinum wire electrode is used.

Typical recordings of cochlear microphonics using two different sound stimulus envelopes are shown (Fig. 1). Column 1 shows a graduated on-off signal and Column 2, a sudden-on, sudden-off type. Line A is a recording direct from the oscillator. Line B shows the modified input wave form, as taken from a sound probe within the external ear canal. Recordings from the round window, tympanic membrane, and skin follow. A 1,000-cycle-per-second free field stimulus at 75 decibels (SPL) was used for the demonstration. This preliminary work was done only in the range of 250 to 1,500 cycles per second due to the frequency limitations of the computer. The cochlear microphonic taken from the tympanic membrane is of a better quality than that recorded from the skin. It should be noted, however, that tracings taken from the tympanic membrane present more of a technical problem in that the animal must be deeply anesthetized and electrode placement is often unstable. The cochlear microphonic obtained from the skin is about 1/1,000 the amplitude of that from the round window and one-tenth that recorded from the tympanic membrane.

More recent work has shown that when the electrode is deeply embedded so that the tip is near the bulla, the microphonic is equivalent to that recorded from the tympanic membrane. In the skin recording in Column 2, line E, it is noted that there is a stimulus synchronized electrical activity superimposed on the cochlear microphonic. This is apparent primarily when using a sudden-on, sudden-off type of tone stimulus. Further work is being done at this time to identify this activity.

Skin-recorded microphonic patterns vary with known types of pathology. The change following plugging of the ear canal with cotton and vaseline is illustrated (Fig. 2). There are no changes in instrumentation between the nonplugged and plugged tracings. The tracing shows a change in amplitude of the cochlear microphonic in the plugged ear.

The microphonic taken from a normal cat ear is shown in Figure 3B. Figure 3C is the pattern taken from the other ear, which had acute purulent otitis media. Note the delayed response time.

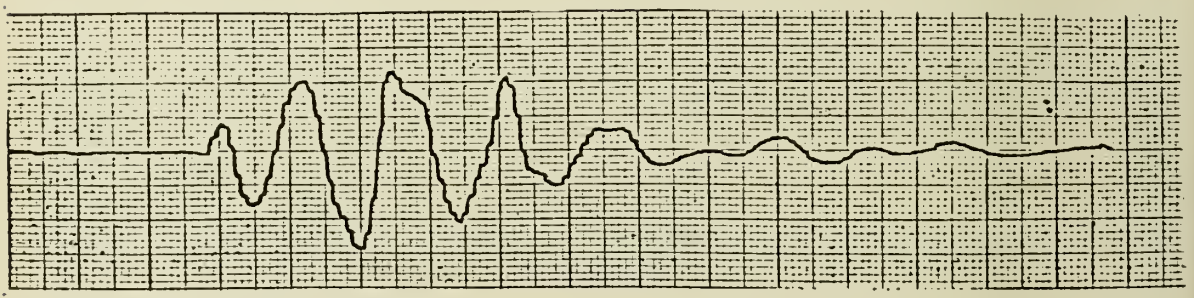
The preoperative microphonic from one ear of another cat is shown in Figure 4A. A tracing taken from the same animal immediately after completing an incus transplantation experiment is shown in Figure 4B. Mechanical impedance and possible surgical cochlear trauma presumably has reduced the microphonic response. Two weeks later, the mechanical impedance and possible cochlear trauma have resolved, and we again record cochlear microphonics (Fig. 4C).

Up to this point we have been interested primarily in gross patterns using sound pressure levels well above the threshold. The next phase of our animal work will be concerned with precise measurements of electrical response and, ultimately, with skin microphonic thresholds.

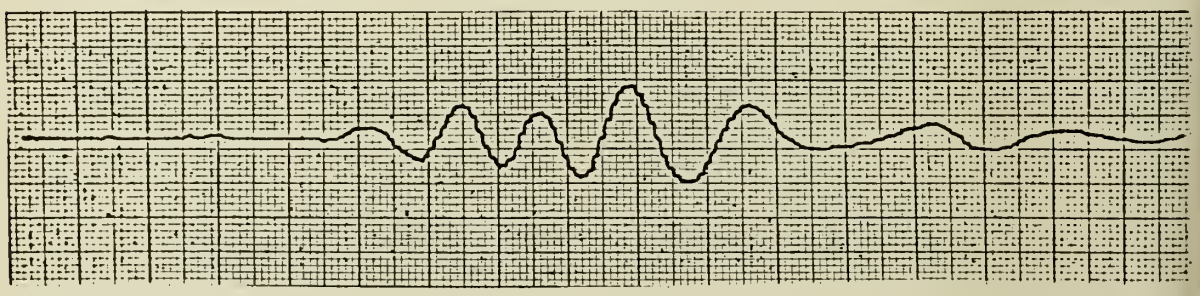
Human application of these methods of hearing analysis is paralleling our experiences with animals, and we are presently solving the problem of a suitable electrode for routine use.

This research was supported by the Louis W. and Maud Hill Family Foundation.

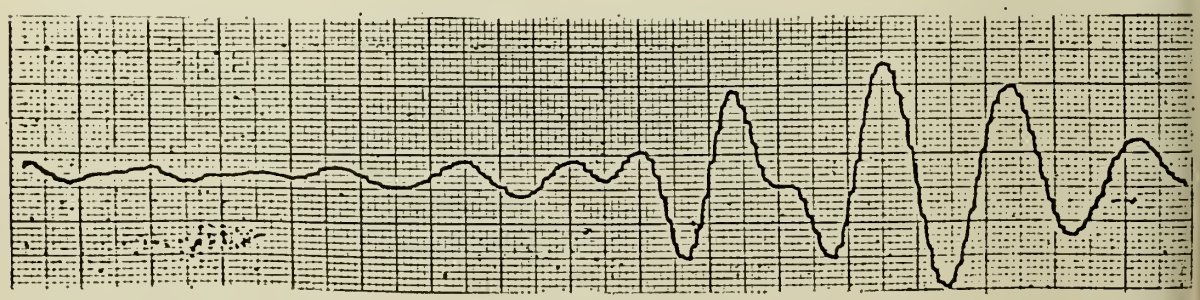
Dr. Boies is the recipient of a special fellowship (NB 1449-01) from the National Institutes of Health.



A Sound probe in external ear canal



B Normal ear (relative gain, × 1)



C Acute otitis media (relative gain, × 5)

Fig. 3. Cochlear microphonic recorded from skin electrodes from a cat with purulent otitis media

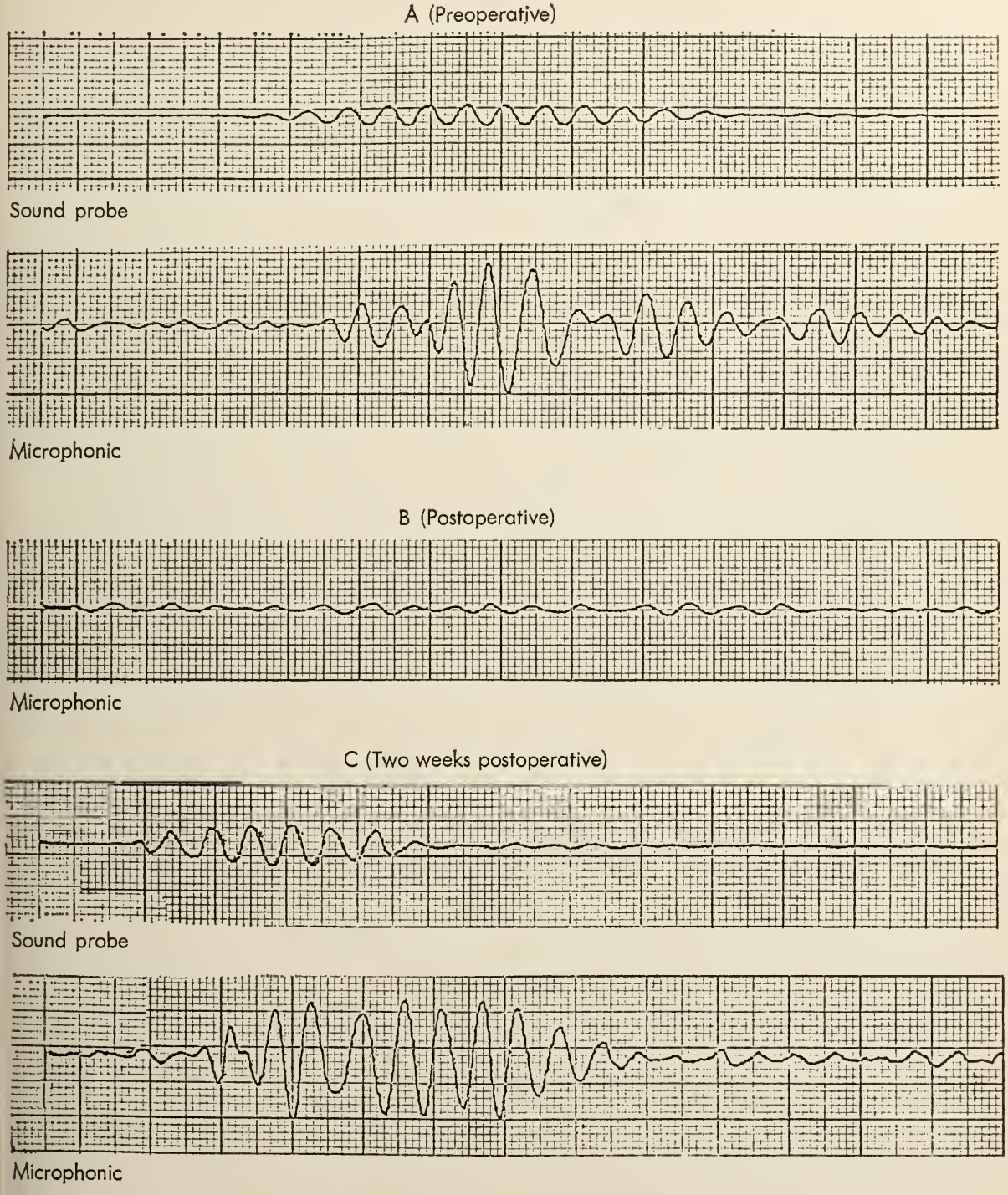


Fig. 4. Skin recorded cochlear microphonic from a cat before and after replacing the incus with an incus from another cat

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Radiotherapy: Art and Science 1965

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WHAT IS RADIOTHERAPY? It may seem unnecessary to define one of the well-established disciplines in clinical medicine, but it is not entirely amiss for I find that somehow radiotherapy has accumulated an air of mystery. This is not entirely surprising because the medical student, intern, and resident rarely have the opportunity to observe at first hand the activities of a radiotherapy department. As the medical trainee enters active clinical practice, the opportunities become even fewer; radiotherapy remains in the minds of most physicians a rather mysterious affair conducted in a mysterious part of a hospital where mysterious men do mysterious things with mysterious equipment. Let us try to dispel this atmosphere of the occult.

Radiotherapy, of course, is what a radiotherapist does, so we might try to characterize the person and thus understand the pursuit. Radiotherapists are physicians who have a special interest in the cancer problem and who use ionizing radiations in the treatment of patients with this disease. They see and examine patients, and they are consulted by their colleagues regarding the relative merits of surgery, radiotherapy, and chemotherapy in the management of patients. When it is agreed that irradiation should be given, the radiotherapist is especially trained to deliver this form of treatment.

Treatment with ionizing radiations entails more than the delivery of a specified dose to the affected part. It involves the day-to-day management of a patient—usually frightened and often in pain—who needs solace, compassionate understanding, and encouragement. The ability to provide this kind of help, coupled with gentle authority and persuasion, is a large part of what is often termed the "art" of medicine. The radiotherapist is called on to provide such support constantly, for there is perhaps no one who requires more of the art of medicine than does the cancer patient. Yet, these qualities have been required in physicians since Hippocratic times and even before. It is nothing new—the only change is that the figure at the bedside is garbed in modern dress. Old though the art may be, it remains one of the most important parts of dealing with the ill. Concerning the cancer patient, it can perhaps be epitomized by stating that the doctor doesn't treat a tumor; he manages a person who has a tumor.

The "science" comes into play in many ways. Always, there is the planning of fields, the calculation of dose, and the plotting of techniques best designed to deliver effective treatment with a minium of early and late side effects in normal structures. Great pains are taken to achieve this end. Effective radiotherapy depends on the relative insensitivity of the normal cell as compared with radiosensitivity of the abnormal, neoplastic cell. If there were not such a "therapeutic ratio" between the one and the other, irradiation could not be used for eradication of neoplasms. Doses lethal to the abnormal tissues would similarly destroy the normal. The problems inherent in the therapeutic ratio concept are magnified and, therefore, perhaps best exemplified when one considers the child with cancer.

Almost every tissue in a child is a collection of rapidly dividing and differentiating cells. The therapeutic ratio in the pediatric patient, therefore, is narrow. Deleterious effects of irradiation can be immediate and obvious; they can be long delayed and extremely subtle. In the first category, bone growth disturbance is an obvious example. Modest doses of irradiation given to the infant will seriously disrupt the normal growth of the part leading to shortening of a limb, asymmetrical growth, or similar orthopedic problems. These can sometimes be avoided entirely, or at least lessened, by the judicious use of lead blocks interposed between the treatment beam and the growth centers, when such shielding does not compromise adequate treatment of the tumor. The long-delayed effects of irradiation, however, are often insidious in onset and difficult to predict. Included in this

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category are oncogenesis and functional impairment. It is now well established that malignant neoplasms can develop in tissue that received prior irradiation. An example is osteogenic sarcoma arising in the facial bone after very heavy irradiation of the orbit for retinoblastoma,[1] a technique that is no longer followed. Less commonly appreciated, perhaps, are the late effects in normal structures leading to their functional derangement. One such patient reported recently developed radiation nephritis with a fulminating malignant hypertension eventuating in death twenty years after irradiation of the abdomen.[2]

These examples of deleterious effects are described not to indicate that irradiation should never be employed in the treatment of malignant disease. Quite the contrary; radiotherapy continues to be one of the most efficient forms of treatment entailing comparatively little morbidity and risk when given carefully by competent personnel. The examples quoted are meant to demonstrate that there is a need to perfect our techniques so as to obtain even better effects in the tumor while lessening still further the reactions in normal structures.

Research into these areas takes many forms. One of the most promising leads has resulted from the investigations of L. H. Gray and others[3,4] of the role of oxygen in the radiation effect. It is now clearly established that oxygen is a semiobligatory cofactor in producing radiation responses in a variety of test systems. Tissues irradiated under low oxygen tension require between two and three times the dose given well-oxygenated structures in order to produce a similar biological response. Since portions of tumors are hypoxic, it is postulated that cells in these areas are much more resistant to radiation effects than the more peripheral cells of the same tumor where the blood supply is better, and therefore the oxygen content higher. The use of hyperbaric chambers, intra-arterial hydrogen peroxide infusions, and similar approaches have evolved to circumvent this problem. For example, at the University of Minnesota, Dr. K. K. N. Charyulu has been studying oxygen gas perfusion of the arterial tree of animal limbs and has demonstrated enhancement of irradiation response in limbs so perfused.[5] Other investigators have attempted to gain a greater tumor response by combining various chemicals with irradiation. Actinomycin D is one of these agents and has been demonstrated in a series of clinical and laboratory observations to produce an enhancement of radiation effects.[6]

So far, the discussion has been limited to irradiation with gamma rays and X-rays. Radiotherapists are interested in the effects of other forms of radiant energy. Included among them are particulate forms such as electrons and heavy particles. These have different properties; for example, they are limited in their range of penetration into absorbers whereas the dose of electromagnetic radiation falls off exponentially. Experiments with heavy charged particles well demonstrate this approach. By heavy charged particle is meant the nucleus of an atom; examples are the hydrogen nucleus (proton) and the helium nucleus (alpha particle). The biological applications of heavy particle irradiation have been under study for some years, notably by J. H. Lawrence and his colleagues at Berkeley.[7]

High-velocity heavy particles have geometric and radiobiological advantages over electromagnetic radiations. The edges of a heavy charged particle beam are extremely sharp with little side scatter into the surrounding medium, allowing far better collimation of the beam. The dose rises steeply in the terminal portion of the beam to a peak, the so-called Bragg peak, after which it drops abruptly within millimeters or less to zero. Thus, the dose at depth is higher than that on the surface, and the dose in tissue deep to the point of interest can be reduced to zero. It is also true that the ionization produced at the Bragg peak is more effective biologically under certain conditions than a similar dose of electromagnetic radiation. It is said to have a higher Relative Biological Effectiveness (RBE). Beams of high RBE are also relatively oxygen independent. Under idealized clinical conditions, not obtainable as yet, beams of this type would be used to deliver high-depth doses to small volumes with minimal radiation to tissues above, below, or beside the locus of interest. These high-depth doses would have a high biological efficiency rendered even higher by reduction of the oxygen effect and, in fractionated treatments, obliteration of cellular recovery after fractionated doses.

These experiments on the effects of heavy particle irradiation have relevance beyond cancer. Astronauts of the future will be subjected to irradiation with particles of various kinds as they traverse interplanetary space. Understanding of the immediate and late effects of irradiation of this kind is an important facet of the research under way in the field of space medicine.[8]

Finally, the radiotherapist seeks to improve his results by inquiries into the nature of cancer itself. In one such study, Dr. Yosh Maruyama of our Division has been investigating the influence

of cell proliferation and cell number on the survival of leukemic mice.[9]

I have endeavored to show that the domain of the radiotherapist ranges wide. It extends from the laboratory and bedside to interstellar space. It embraces practices that are among the most honored and ancient in medicine, along with techniques that are as modern as tomorrow.

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Continued from page 235

ACUTE RHEUMATIC FEVER: A NEW LOOK

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A Neglected Roentgen Diagnostic Feature of Ruptured Aneurysms of the Abdominal Aorta

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Ruptured aneurysms of the abdominal aorta have many characteristic roentgenological features that have been described in detail by numerous authors,[1,2] and no attempt will be made to review them here. The purpose of this presentation is to describe an often overlooked roentgen sign which either may serve as an additional X-ray finding in a ruptured aneurysm of the abdominal aorta or, on some occasions, may provide the only conclusive evidence on a plain film of the abdomen of this vascular catastrophe.

A review of anteroposterior films of the abdomen of five consecutive verified cases of ruptured aneurysms of the abdominal aorta revealed a triangular shaped soft-tissue density along the left side of the pelvic cavity (Fig. 1). The medial border of this density runs diagonally from about the inferior border of the sacroiliac joint to the ischial spine along the expected position of the pelvic portion of the psoas muscle. The lateral border of the soft-tissue density conforms to the inner aspect of the iliac bone and sometimes overlaps the bone itself. This density is slightly above the expected location of the obturator internus muscle. The latter can sometimes be visualized on a good roentgenogram of the pelvis as described by Levene and Kaufman.[3] In most routine films of the abdomen, however, the shadow of the obturator internus muscle seldom can be identified with a reasonable degree of certainty. The above described soft-tissue density represents the downward extension of a retroperitoneal hematoma from a ruptured aneurysm along the pelvic portion of the psoas muscle. Other shadows that must be differentiated from this density are: fecal material in the sigmoid colon, free fluid in the peritoneal cavity, hypertrophy of the psoas or obturator internus muscles, pelvic abscess or neoplastic process (intra- or retroperitoneal), and, finally, retroperitoneal hematoma or fluid (urine) secondary to a trauma with or without skeletal fracture. History, clinical findings, and the roentgenological features of the soft-tissue density in the lesser pelvis seen in the above conditions are sufficient to permit a correct interpretation. Furthermore, the straight medial



Fig. 1. Arrows point to the large pelvic retroperitoneal hematoma. Note also obliteration of the left psoas shadow and absence of calcification in the abdominal aorta.

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Fig. 2. [A] Solid arrows point to the injected retroperitoneal fluid in a cadaver. Broken arrows point to the extension of the same fluid in the floor of the lesser pelvis. [B] Appearance of the pelvis before retroperitoneal injection.

border of the soft-tissue density of the retroperitoneal hematoma along the pelvic segment of the psoas muscle is the striking feature of ruptured aortic aneurysm.

Experimentally, one can reproduce the soft-tissue density of a retroperitoneal hemorrhage on a simple film of the abdomen by injecting fluid into the retroperitoneal space or directly into the psoas muscle. This was demonstrated in the left lesser pelvis (Fig. 2A) in a fresh cadaver after injection of 300 cc. of water into the left retroperitoneal space. The change can be readily appreciated after comparison with the film of the cadaver taken prior to retroperitoneal injection (Fig. 2B). During the same experiment, X-rays of the pelvis were made after each injection of 50 cc. of fluid until a total volume of 500 cc. was reached. It was found that the size of the soft-tissue density in the lesser pelvis on the film did not increase in proportion to the amount of fluid injected. This was due to gradual spread of water into the adjacent tissue and also to escape of fluid from the retroperitoneal space into the peritoneal cavity. It is obvious that the use of the cadaver, where there is no dynamic interplay of the anatomic structures and interspaces, has many limitations. Furthermore, the use of water, which is more diffusible than blood, can hardly be expected to duplicate the actual presence of blood in the retroperitoneal space under continuously augmenting pressure from the lumen of an actively leaking aorta. In the latter event, there is increased tension and an actual tamponade effect in the retroperitoneal

SUMMARY OF ROENTGENOLOGICAL FINDINGS IN FIVE CASES OF RUPTURED ABDOMINAL ANEURYSMS

Patient's initials, age, and presenting symptoms	*Palpable abdominal mass*	*Visible aortic calcifications*	*Psoas muscle obliteration*	*Demonstrated pelvic retroperitoneal hematoma*
C.H., 78 years; pain in left lumbar region since morning of the day of admission	No	None in antero-posterior but present in lateral view	Yes	Yes
C.H., 64 years; sudden onset of left flank pain with radiation to left testicle	Yes	Yes	No	Yes
A.H., 67 years; epigastric pain of twenty-four hours' duration	Questionable	Yes	No	Yes
L.Y., 68 years; sudden onset of severe back pain two hours prior to admission	Yes	Yes	Yes	Yes
O.K., 57 years; severe abdominal pain occasioned by bending forward three hours before admission	No	Barely visible or none	Yes	Yes

spaee that eannot be adequately reprodueed in a eadaver with injection of the readily diffusible water. The experiment would have been more satisfactory if a catheter were inserted into the retroperitoneal space at the level of the kidneys and if blood instead of water had been injected.

DISCUSSION

It should be noted that the mere obliteration of the psoas outline does not neeessarily indicate rupture of an aneurysm when the latter is suggested clinieally or roentgenologically. Ryan and associates,[4] in a review of 191 cases of proved abdominal aneurysms (without rupture), found obliteration of the psoas shadow in 12 cases. However, when an abdominal aneurysm is demonstrated with the obliteration of the psoas muscle, the presence of the above described soft-tissue density in the left lesser pelvis should serve as conclusive evidence of its rupture. In two of the five cases of ruptured abdominal aneurysms presented here, the psoas muscle outlines were not obliterated at all. A summary of the roentgenological findings is given in the table.

Grayson and Kennedy,[5] in a report of three cases of ruptured aneurysms of the abdominal aorta, presented roentgenological evidence of the extension of a hematoma into the pelvis in one ease. In a seeond case, they demonstrated ealcifieation in a hematoma seeondary to an old ruptured aneurysm. This was shown not only in the retroperitoneal space of the pelvis but in the retroperitoneal tissue of a hernia as well. Klinefelter[6] described a case in which the hematoma from a ruptured abdominal aneurysm had produced a filling defeet in the reetum. This was recognized during a barium enema examination. These are the only referenees which were found in a review of literature concerning pelvic manifestation of ruptured aneurysms of the abdominal aorta.

SUMMARY

Attention has been drawn to the presence of a triangular shaped soft-tissue density in the left lesser pelvis in cases of ruptured aneurysms of the abdominal aorta. This is believed to be due to the extension of a hemorrhage into the pelvic portion of the retroperitoneal space. When there is clinical or roentgenologieal evidenee of an aneurysm, the presence of this soft-tissue density should alert one to the probability of a rupture. This may prove to be of more significance than the obliteration of the psoas muscle, which has been known to occur in some abdominal aneurysms without rupture.

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END — St. Paul–Ramsey Hospital Symposium

A Transsexual Research Project at the University of Minnesota Medical School

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Newspaper accounts of the "sex changing" project at the University of Minnesota appeared in December 1966. Since then, numerous physicians have inquired about the cardinal symptoms of transsexualism and the criteria for referral. The purpose of this brief report is to describe the syndrome and outline the research project.

THE SYNDROME

Transsexualism involves a reversal of the sexual role from a psychological standpoint, that is, the male feels that he is a female in a man's body. The female transsexual regards herself in the opposite way. Incidence is unknown, but it is thought to occur more frequently in males. Characteristically, the patient ardently desires a surgical change of sex insofar as this is possible, and this is a hallmark of the syndrome. The patient often has a loathing for his genitalia and dislikes touching the area while bathing, for example. From the standpoint of anatomy, physiology, and chromosome and endocrine studies, the male transsexual is male and the female transsexual, female. No related abnormalities are ordinarily found.

Transsexuals usually dress in the clothing of the opposite sex, privately or in public, or wish to. Such males may come to the attention of the police for cross-dressing in public. If the female cross-dresses in public, she rarely is apprehended. Dressed in slacks and other mannish attire, she attracts little attention. However, a man does, but many appear so feminine that detection by a police officer is accidental. A common example is that of the male transsexual, dressed as a woman, when stopped for a minor traffic violation. In examining the driver's license, the officer finds that it describes a male, but the driver appears to be a woman and is arrested for fraudulent use of the license. Subsequent explanation and physical inspection at the police station reveal the truth.

These people are not psychotic or otherwise mentally ill, unless the desire to be a member of the opposite sex is construed as a symptom of mental illness. They are extremely unhappy, distressed persons, however, and understandably have difficulty achieving a satisfactory social adjustment.

The question of homosexuality in these cases is an interesting one and points up the dilemma these patients face. Their wishes and sexual fantasies are concerned with falling in love with and marrying a man, having coitus with him in the role of a female, and adopting children to raise and mother. These patients readily admit that they are homosexual if judged by their anatomical sex. However, the transsexual views himself as heterosexual, not homosexual. "Psychologically I am a female. This is the way I feel. I want to love a man as a female." The transsexual usually reports that he is disgusted by any thought of relations with a female since this, to him, would be homosexual in nature.

Attention of the American public was first directed to this condition by the Christine Jorgensen case in 1952. Jorgensen was a former U.S. soldier who was operated on in Denmark and returned to this country as a female. She then went on the stage and achieved considerable notoriety. This had two effects: the public became aware that such a situation existed and, as a backlash of the notoriety, Scandinavian surgeons stopped operating on foreign patients.

Most American transsexuals who have had operations during the past decade have gone to surgeons in Casablanca or Mexico, but a few have had operations in the United States without public notice. Johns Hopkins Medical School announced its transsexual project in the fall of 1966.

Etiology. The etiology of transsexualism is not

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understood. As mentioned before, there does not appear to be a genetic factor involved. The developmental history, as given by the parents, indicates that the child was "different" from the early years of life. It is characteristic that this little boy wanted to play with dolls and that he could not be interested in the activities of boys. It is routine to hear parents tell that their son wanted to wear dresses, use his mother's lipstick, help his mother in the kitchen, care for younger babies born into the family, and do handiwork, such as sewing. It is the rule to hear the father say that, try as he might, he could not interest his son in hunting, fishing, boys' games, or other male pursuits. Whatever the etiological factors are, they apparently occur between birth and about age 3. This suggests some type of learning or conditioning process. Various theories have been suggested, ranging from "imprinting" (although this phenomenon has not been demonstrated to occur in human beings) to factors such as the parents' desire for a girl instead of a boy and then treating the baby as a girl in subtle ways. The etiology, however, is not known from a factual standpoint.

Treatment. Numerous therapies have been attempted, ranging from psychoanalysis through drugs to electroshock therapy. None has shown any benefit and all are regarded as worthless by physicians acquainted with the condition. There are two possible therapeutic approaches:

1] Change the mind to fit the body, that is, change the female psychological role to a masculine one in order to conform to the anatomical sex. As mentioned, there is no treatment known to accomplish this.

2] Change the body to fit the mind insofar as this is possible. This is the focus of the present research project.

DESCRIPTION OF THE RESEARCH PROJECT

Our attention was first drawn to this clinical problem by a transsexual patient admitted to the psychiatric service at the University Hospitals in December 1964. Impressed with the man's suffering and his sincerity in trying to obtain surgery, a series of discussions and meetings were held, finally leading to the inauguration of the research project. We received outstanding cooperation from members of the law school faculty and several members of the private legal community; the hospital, university, and medical school administrations; the medical school-hospital committee; the committee on human experimentation; and other medical school departments. It was truly a lengthy process—in fact, several years in duration—before we were ready to start. During the planning phase, two members of the gender committee visited New York City and examined three transsexual patients who had had operations in Casablanca. They were impressed with the excellence of the surgery and with the good social and emotional adjustments reported by the patients.

If one discusses treatment possibilities with a transsexual patient, one quickly finds that he is convinced that his salvation lies in surgical change. The current research project puts this assumption to the test. In brief, the plan is to operate on a minimum of 20 male transsexuals and then do a ten-year follow-up study to determine the postoperative social, emotional, sexual, and economic adjustments the patients make living as females.

Selection criteria. Like any admission to the University of Minnesota Hospitals, the patients must be referred by a physician. At the present time, no one under 21 years of age is considered, nor are there plans to operate on females. No psychotic or mentally defective person will be accepted because of the impossibility of signing a valid operative permit. Nonresidents of Minnesota are not considered unless they are private patients, that is, they participate at no expense to the state of Minnesota, but residents are admitted under the same financial provisions that apply for other patients at the University of Minnesota Hospitals. No private patients will be accepted from countries other than the United States and Canada because of the follow-up difficulties that would be involved with a foreign national.

After the publicity appeared, hundreds of requests for surgery arrived from people in all sections of the United States and from foreign countries, including Europe, South America, Guam, India, and Asia. The youngest correspondent stated he was 12 years old and the oldest, 73. Males by far outnumbered the females.

Initial study. If these criteria are satisfied, the patient is seen first on an outpatient basis to acquaint him with the research project and to form a tentative opinion about diagnosis and mental status. Unless some obvious reason is found to disqualify him, arrangements are made for admission to the psychiatric service for a two-week period of study. These studies consist of history (also obtained from parents, if available), physical and neurological examinations, routine and indicated laboratory work, and extensive psychiatric interviewing and psychological testing. Chromosome counts and 17-ketosteroid determinations are done. The patient

is recorded on video tape as he gives a brief version of his history and the reasons for wanting surgery. At the conclusion of this period of hospital study, the patient meets the gender committee which is composed of a psychologist, surgeon, obstetrician-gynecologist, hospital administrator, psychiatric nurse, and psychiatrist. This committee considers the assembled data and votes to include or exclude the patient from the research study according to two primary considerations: Is the diagnosis correct? and is the patient mentally competent? The committee does not attempt to assay factors that are mainly guesswork, such as what kind of female the person may become, and the like. However, the patient usually is asked to appear before the committee dressed as a female to form an idea about feminine appearance.

If the person still desires surgery and is accepted by the committee, there is a commitment to operate. Thus, in this sense of the word, his long quest for surgery is at an end. Knowing he can have an operation, the patient now can arrive at a more objective decision about the surgery.

Estrogen phase. The patient is then placed on relatively high-dosage estrogen therapy for the next six months. This acts as an endocrine "sedative" in that the person feels less tense. It produces some enlargements of breasts and also feminine contouring, and it suppresses sexual drive and erections, results which are welcomed by the patient. But most important, this phase provides a six-month waiting period during which the person can consider and reconsider whether he wishes surgery. At the end of the six months, the person is readmitted for final evaluation and is operated on, if this is his decision.

During this six-month period, the patient is required to obtain a private attorney to represent him, to examine the operative permit with him, and to be ready to help with the legal matters involved, such as change of name.

Operative phase. The surgery consists first of circumscribing the skin of the phallus just beneath the corona of the glans. A tube of penile skin is then developed by dissecting the skin off the shaft of the penis. The entire shaft of the penis plus the crura are removed through the same distal penile incision. Both testes are removed through this same opening. After broadly dilating this opening, the rectum is peeled back off the prostate and seminal vesicles, creating a cavity such as in a radical perineal prostatectomy. The prostate and seminal vesicles, however, are not removed. The vagina and labial folds are fashioned from the intact penile and scrotal skin. The urethra is brought out in the typical female position under the pubic symphysis between the newly created labia.

A silicone rubber vaginal mold is inserted for one week. After that, the plug is removed but the patient is taught to replace it daily for several hours to keep the vaginal barrel patent.

The operative phase lasts three to four weeks, depending upon convalescence required. Toward the end of this phase, nurses help the patient, if need be, with the feminine role—how to apply lipstick and eye shadow, sit with legs crossed appropriately, and the like. Most patients require little training of this type because usually they will have had experience appearing in public situations that require feminine behavior and appearance.

As of this writing, three patients have completed the operative phase.

Follow-up phase. It is planned that patients will be interviewed and restudied on an outpatient basis every six months for a ten-year period. The follow-up will examine the social, emotional, sexual, and economic adjustments the patients actually make. From this, we hope to determine whether such surgery is a worthwhile approach to an otherwise untreatable condition.

Credit for the term, transsexualism, usually is given to Dr. Harry Benjamin who, over the past several decades, has been the person most influential in bringing this topic to the attention of the medical profession in the United States.

The authors are indebted to Drs. Harry Benjamin, Wardell Pomeroy, and Leo Wollman, New York City, for making it possible to examine the three transsexual patients who had their operations in Casablanca.

Mediastinal Abscess of Subdiaphragmatic Origin

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MEDIASTINAL ABSCESS secondary to subdiaphragmatic pathology is unusual. Before the use of antibiotics, infection in the mediastinum occurred from extension of the inflammation along the fascial planes in the head and neck. Although this has been eliminated, infection still occurs from perforation of the esophagus as result of trauma, ulceration, or neoplasia. The present case deals with the development of mediastinal abscess as a complication of perforated duodenal ulcer. Although Buist[1] reported a patient who developed such an abscess secondary to a liver abscess,[2] a search of the English literature failed to disclose other patients who had had a mediastinal abscess as a complication of subdiaphragmatic disease.

CASE REPORT

A 45-year-old white male alcoholic was admitted to West Side Veterans Administration Hospital, Chicago, Ill., on Oct. 21, 1964, with a twenty-four-hour history of severe generalized abdominal pain and vomiting. The findings were consistent with an acute surgical abdomen and on Oct. 22, 1964, a laparotomy was performed. A perforated duodenal ulcer was found, the opening closed, and a gastrostomy performed. Numerous postoperative complications developed, the most pertinent of which were wound dehiscence and a pelvic abscess which drained spontaneously per rectum on the eleventh postoperative day. In the interim, the patient was found to have pulmonary tuberculosis, and for this reason he was transferred to Veterans Administration Hospital, Hines, Ill., for further treatment. Upon admission to Hines, an X-ray of the chest and upper abdomen revealed a fluid level beneath the left diaphragm (Fig. 1). Gastrografin swallow disclosed that the stomach was displaced medially and downward by a collection of air and fluid in the left subphrenic space (Fig. 2). On the fourteenth postoperative day, under local anesthesia, a left subdiaphragmatic abscess was drained through the lateral approach in the tenth intercostal space at the posterior axillary line. On the day following this procedure, the patient was noted to have a fluid level in the mediastinum. It was felt that this might have occurred by direct extension. Since the subphrenic abscess was adequately drained,



Fig. 1. Subphrenic abscess, anteroposterior view

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Fig. 2. Gastrografin swallow showing stomach being pushed medially and down by subphrenic abscess

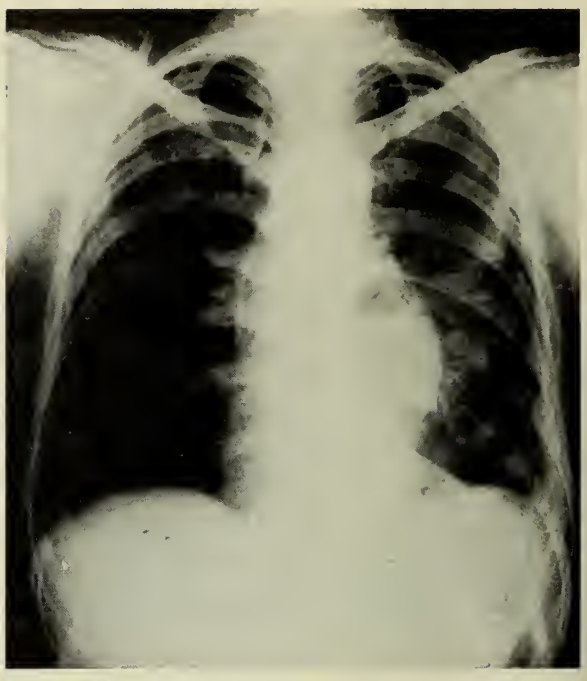


Fig. 3. Mediastinal abscess on the day it was drained, before surgery

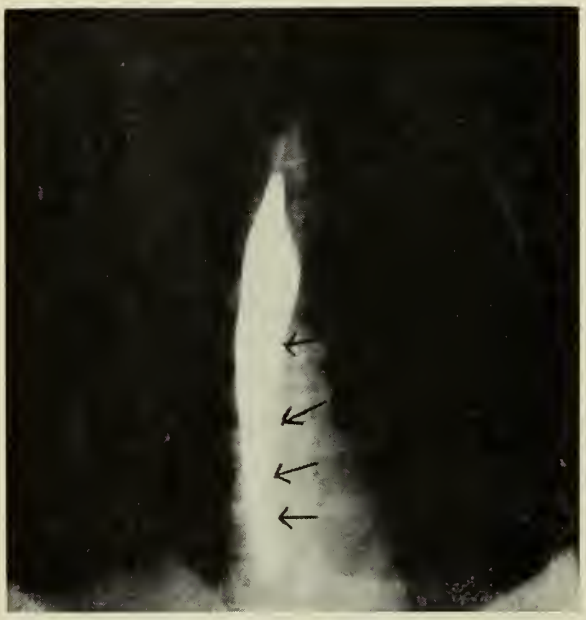


Fig. 4. Esophagogram showing the mediastinal abscess displacing the esophagus to the right

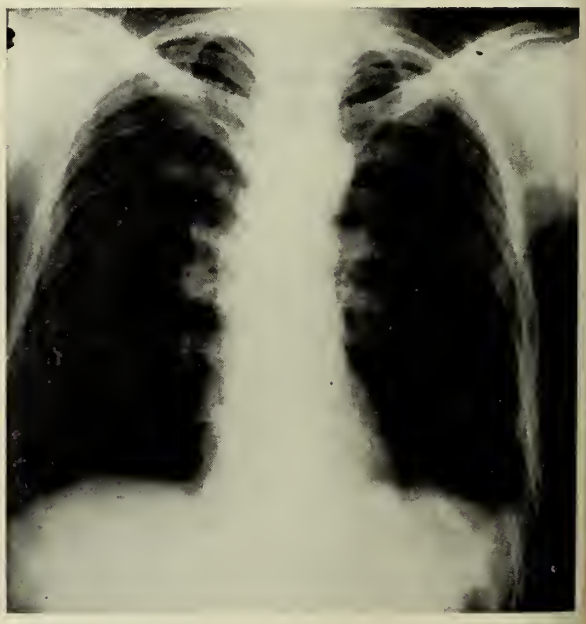


Fig. 5. Chest X-ray taken about two weeks following drainage of mediastinal abscess

it was hoped that the mediastinal abscess might evacuate through the same route. For this reason and because of an acute, toxic brain syndrome, a course of antibiotics was instituted. Improvement of both entities was manifest clinically during the subsequent week. However, his fever recurred and serial X-rays disclosed enlargement of the mass (Figs. 3 and 4). Therefore, on the thirty-second day following the closure of the duodenal ulcer, under local anesthesia and utilizing an extrapleural approach, the mediastinal abcess was drained by resecting the posterior portion of the tenth rib. Culture of the pus revealed Streptococcus viridans. His postoperative course was uneventful and he has remained well (Fig. 5).

DISCUSSION AND CONCLUSION

The paucity of reports of mediastinal abscess resulting from disease below the diaphragm prompted a report of this case. As a rule, the diaphragm provides an effective barrier to the spread of infection from the abdomen to the chest. In this patient, resistance to the spread of infection was probably lessened by poor nutritional status from chronic alcoholism and pulmonary tuberculosis.

Spread of infection is usually ascribed to one or more of three routes: hematogenous, lymphatic, or direct extension. Because of the proximity of the two lesions and the absence of other abscesses, it would appear that direct extension and ascending lymphatic spread, or both, occurred in this case. Webb[2] describes a retrovisceral space lying between the esophagus and prevertebral fascia which communicates with the retroperitoneal area via the esophageal hiatus. Since abdominal esophagus is covered anteriorly and to the left by peritoneum, it is conceivable that the subphrenic abscess under pressure ruptured the peritoneal barrier and ascended into the mediastinum. However, ascending lymphatic spread could also be postulated as a logical pathway. Although the fluid in the mediastinum was first noted the day following drainage of the subphrenic abscess, no doubt the mediastinal abscess had been developing before this time.

The posterior extrapleural approach to the mediastinum, as described by Lindskog,[3] was found to be quite satisfactory. It is not technically difficult and can be performed under local anesthesia, and adequate drainage can be obtained when the abscess is accurately localized. Thus, the pleural space is not entered and an empyema is avoided.

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A Promising Procedure for the Treatment of Urinary Incontinence in the Male

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TOTAL URINARY INCONTINENCE following prostatectomy is a most undesirable complication. It is relatively rare in occurence, but this provides little comfort for the unfortunate patient who must cope with its problems. The literature is replete with ingenious operative techniques which promisc only an occasional success. In 1961 Dr. Berry[1] reported a new surgical procedure for the control of urinary incontinence in the male (Figs. 1 and 2). An acrylic prosthesis with a silastic balloon is implanted through a midline perineal incision to saddle the urethra. Fixed in position with wire sutures through the ascending rami of the ischium and pubis, the prosthesis creates an increase in urethral resistance sufficient to achieve control, yet allow a free flowing stream during micturition. (Figs. 3A, 3B, 4A, and 4B).

Under the tutelage of Dr. Berry, we began using his procedure in January 1965. Since that time, we have performed 9 Berry procedures on 8 patients with histories of incontinence, (Table 1), from six months to over ten years duration, following operative procedures at our hospital and others. Five had incontinence following transurethral resection of the prostate, 2 following radical perineal prostatectomy for carcinoma, and 1 following radical retropubic prostatectomy for carcinoma. Seven patients had an excellent immediate result and there was 1 technical failure. One patient suddenly became incontinent ten months later, following intercourse. His prosthesis was replaced and the result was excellent. The remaining patients have been followed up to one and a half years. Two have developed minimal stress incontinence and 1 man suffered severe incontinence after a paroxysm of coughing. This suggests that in time there may be a failure of complete control in some patients after primary success.

The silastic balloon has been designed for expansion to reestablish pressure if the prosthesis has slipped. With the simple technique and low morbidity, we have not hesitated to replace the entire prosthesis.

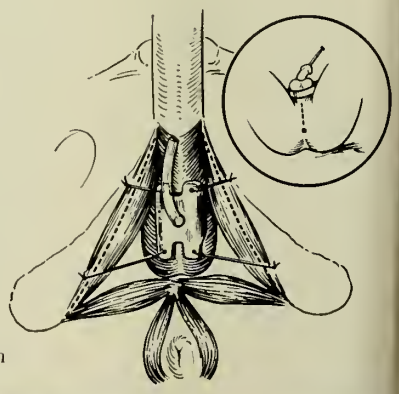


Fig. 1. *(Right)* Drawing of surgical procedure

Fig. 2. *(Below)* Berry prosthesis and silastic balloon



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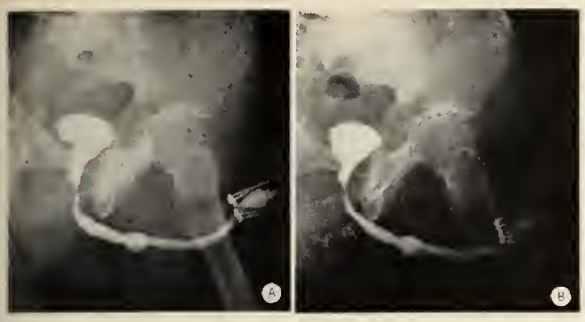


Fig. 3A. Preoperative urethrogram (B) Postoperative urethrogram



Fig. 4A. Preoperative urethrogram (B) Postoperative urethrogram

Dr. Berry[2] reports a success rate of about 50% for his procedure all over the country. Better technique and procedure may evolve, but we are grateful to have a reasonable chance of success to offer our patients now.

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INCONTINENCE

Case	*Age*	*Etiology*	*Duration (months)*	*Degree*	*Immediate results*	*Late results*	*Remarks*
1. R.K.	53	Transurethral resection	106	Severe stress	Excellent	Good at 16 months	Occasional stress incontinence
2. S.H.	71	Transurethral resection	9	Total	Excellent	Excellent at 16 months	
3. J.R.	68	Transurethral resection	6	Total	Excellent	Good at 16 months	Stress incontinence in cold weather
4. T.P.	69	Transurethral resection	11	Total	Excellent	10 months, later became incontinent	Prosthesis replaced with excellent result
5. V.L.	73	Radical perineal prostatectomy	51	Total	Failure		Not improved with balloon expansion
6. M.M.	71	Radical retropubic prostatectomy	37	Severe stress	Excellent	Fair at 9 months	Stress incontinence after 7 months following cough
7. E.D.	63	Transurethral resection	61	Total	Excellent	Excellent at 4 months	Pain from balloon stem
8. M.D.	72	Radical perineal prostatectomy	33	Total	Excellent	Excellent at 3 months	

John A. Anderson, M.D.

LEWIS W. WANNAMAKER, M.D.
Minneapolis



Born in South Dakota, educated in the Midwest, and nurtured in the McQuarrie tradition, John A. Anderson has brought a wide range of talents and interests to the position of head of the Department of Pediatries at the University of Minnesota.

Son of Lars and Minnie Anderson, he was born in Sioux Falls and attended Washington High School and, for two years, the University of South Dakota. During the twelve years after transferring to the University of Minnesota, he accumulated four degrees, a B.S. in 1930, an M.B. in 1933, an M.D. in 1934, and a Ph.D. in physiology and pediatries in 1940. The traditional separation of the Twin Cities must not have been as complete as some historical accounts would lead us to believe since, in 1934, he married Albertine Agatha Arth from St. Paul. In subsequent years, they have adopted two children, Judith Kay, now 22 and recently married to Gerald Gustafson, and Robert John, now 17.

After an inspiring introduction to the basic sciences in the medical curriculum, as presented by those intellectual giants, the Paul Bunyans of the University of Minnesota's rise to national medical prominance—Bell, Jackson, Downey, and Scammon—John Anderson admits that he reacted strongly when, as a junior student, he suddenly was brought face to face with the empiricism of clinical medicine. It seems likely that this experience might have driven him back to the preclinical sciences as a career had it not been for the perplexingly complex but stimulating lectures of Hilding Berglund, professor of medicine, and Irvine McQuarrie, professor of pediatries, who posed difficult questions rather than provided pat answers—an educational technique which still fires the scholar and sends the mere student scurrying back to his synopses.

During his medical school days, John Anderson took an elective with Dr. J. C. McKinley in the Department of Neurology and Psychiatry. This "extracurricular elective" called for a minute dissection of the human brain, a pursuit which must have been well suited to Dr. Anderson's meticulous nature and which may be the foundation for his long-standing interest in neurology. It is also of note that early in his career John Anderson spent three months as an intern on the obstetrical service of the Minneapolis General Hospital, now the Hennepin County General Hospital. This orientation may be responsible for his particular concern for problems in the perinatal area.

Only after these brief excursions into other disciplines did John Anderson settle down with the field of pediatries as his home, accepting the McQuarrie philosophy that pediatries encompasses all of the medical and biological sciences and therefore must relate to all of these disciplines. As a pediatric resident, he found time to work, under Dr. McQuarrie's supervision, with

LEWIS W. WANNAMAKER *is with the Department of Pediatrics, University of Minnesota Medical School, Minneapolis.*

Mildred Ziegler in the laboratory on metabolic problems. His first paper, which appeared in the *Journal of Nutrition* in January 1936 and on which he was coauthor with Dr. McQuarrie and W. H. Thompson, was concerned with excessive ingestion of sodium and potassium salts in diabetic children and examined the effects of such ingestion on blood pressure and carbohydrate metabolism. Another early paper which appeared in the *Proceedings of the Society for Experimental Biology and Medicine*, published jointly with A. V. Stoesser and Dr. McQuarrie, explored the effects of various diets, cholesterol, and choline on lipids in the rat liver.

In contrast to most young men of his day, who went east to Baltimore or Boston to broaden their medical horizons, John Anderson went west to Denver. There he served as a fellow, under Commonwealth Foundation support, in Al Washburn's Child Research Council. This period undoubtedly added a new dimension to his experience and sharpened his appreciation of longitudinal studies in pediatrics, a concept and approach which he was to bring to bear in later developments in his own department at Minnesota.

John Anderson returned to Minnesota in 1937 and served as an instructor until 1940 at which time he received his Ph.D. degree in physiology and pediatrics. Two papers in the *Proceedings of the Society for Experimental Biology and Medicine* reflect his interests at that time in the effects of relative humidity on insensible weight loss and on skin and rectal temperatures of newborn infants. For three years he stayed on as assistant professor and studied the antagonism of pitressin and adrenal cortical extract in human diabetes insipidus and the effects of desoxycorticosterone acetate on water exchange and on the water and electrolyte content of tissues, particularly the brain.

Following the McQuarrie pattern of up and out—to fill the rapidly developing posts in academic pediatrics throughout the West—John Anderson left the University of Minnesota in 1943 to become, at the age of 35, the chairman of the Department of Pediatrics at the University of Utah. During the next six years, while serving in this position, he developed a strong interest in the clinical, epidemiologic, therapeutic, and experimental aspects of poliomyelitis, a disease which was terrorizing many parts of the country, including Utah, at that time. This interest resulted in a series of papers on this and, subsequently, other diseases of viral etiology.

In 1949, John Anderson moved to Stanford University as head of the Department of Pediatrics. There, he played a key role in the development and expansion of that medical school before the move to its present location in Palo Alto. A study of the effect of the Lansing strain of poliomyelitis virus on phosphorus metabolism of monkey brain, published in the *Proceedings of Experimental Biology and Medicine* in 1950, led to a continuing interest in the metabolism of phosphorus in the central nervous system. This work was elegantly and eruditely summarized in his contribution to *Essays on Pediatrics*, a symposium in honor of Irvine McQuarrie held in 1954.

The continuing high regard with which Dr. McQuarrie, Dean Harold S. Diehl, and other influential faculty members at Minnesota held John Anderson was brought to light shortly thereafter in 1955 when he was selected from a large field of fair-haired pediatric sons of Minnesota and other candidates to take over the Department of Pediatrics at his own alma mater.

Over the years, Dr. Anderson's abilities have received recognition in other ways. He has been elected to a number of medical societies and has served as president of the Intermountain Pediatric Society, the Northern California Pediatric Society, and the Western Society for Pediatric Research, as council member of the Society for Pediatric Research, as chairman of the council of the American Pediatric Society, as vice-president of the Physician's Council, as a board member of the American Board of Pediatrics, as a member of the editorial board of *Pediatrics* and of *Advances in Pediatrics*, and as editor of the *American Lecture Series in Pediatrics*.

Precise in speech and manner, John Anderson is, nevertheless, known for his camaraderie with colleagues and for his warm rapport with children, whose company he enjoys. He has demonstrated his skill as a craftsman in his home workshop. He is venturesome and enjoys fishing and exploring in the North Woods. He has a volatile sense of humor and is recognized among his friends for his poetry writing, with an apt and witty verse for every occasion. Among the house staff, he is known for the complicated metabolic diagrams which he spontaneously draws at seminars and for his comments on grand rounds which range from astute clinical observations to tangential esoteric speculations. Among the members of his department he is known for the sessions in his office which, while rarely resulting in a solution of the practical problem at hand, usually lead to a lively intellectual discourse on a variety of unrelated but stimulating subjects.

When John Anderson returned to Minnesota for the second time, he brought with him twelve

years of administrative experience gained as departmental chairman at Utah and at Stanford. He was quick to accept the challenge of increasing support for medical research to enlarge, strengthen, and broaden the department at Minnesota. Particular emphasis has been placed on developing ties with other departments in the medical school; on interdisciplinary cooperation in the areas of neurology, pathology, virology, and perinatal physiology; and on rebuilding the areas of endocrinology and metabolism following Dr. McQuarrie's departure. John Anderson has recognized the importance of horizontal relationships, a concept which he has managed to adapt and transfer from the flat structures of California to the top floor of the Mayo Memorial Building. His research interests since his return to Minnesota have been in blood ammonia levels and in 5-hydroxytryptamine and phenylalanine metabolism.

While following the McQuarrie tradition, John Anderson has gradually molded the Department of Pediatrics along new lines of general emphasis. Dr. McQuarrie, trained in internal medicine, was later grafted onto pediatrics. He was a genial showman, the Robert Ripley of pediatrics, who capitalized intellectually rather than monetarily on his collection of freaks. He was concerned with the task of bringing pediatrics to a level of clinical investigation on a par or beyond that of internal medicine. John Anderson has equally promulgated the importance of applying the tools and concepts of basic science to the solution of clinical problems but has further recognized the unique role of pediatrics as a developmental science. This is witnessed by his absorbing concern with the Child Development Study, a cooperative venture involving several departments and a number of institutions which brings together his interests in perinatal physiology, in neurology, and in longitudinal studies. More recently, he has been a prime promoter of the new NIH Institute of Child Health and Human Development, which should provide additional opportunities for development-oriented studies both at Minnesota and elsewhere in the country. One other trend can be detected since Dr. Anderson arrived at Minnesota. He has taken advantage of burgeoning federal support to encourage young men to stay at Minnesota and develop new areas rather than move on to strengthen or head new departments in other schools. This has resulted in a steady expansion of the department to its present size and position in academic pediatrics. Present trends in federal and legislative support and in university administration will present new challenges to John A. Anderson, problems of maintaining strength and excellence which may be equally as formidable as the hurdles of building and expanding a department. He brings seasoned experience and an unfaltering spirit of commitment and excitement to the tasks which lie ahead.

Book Reviews . . .

Prevention of Ischemic Heart Disease: Principles and Practice

edited by WILHELM RAAB, M.D., *466 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $25.50*

Proceedings of the First International Conference on Preventive Cardiology at the University of Vermont, held in August 1964, provide most of the papers in this book. Authors are from many countries.

There are four sections: The first called "Pathophysiological Fundamentals" deals largely with studies of catecholamines in various situations, especially their variation under emotional and other neurogenic stress.

The second section, "Epidemiological Data," considers population differences, effects of exercise, diet, weight, socioeconomic status upon morbidity, and mortality rates of coronary sclerosis. Considerable attention is given to hypercholesterolemia and the possible effects of diets.

In section three, "Preventive Measures," there are several papers on exercise. A number of authors advise exercise up to exhaustion and claim prophylactic and therapeutic results in angina pectoris and myocardial infarction. A report from Israel states that rate of exercise up to extreme exertion has a beneficial effect upon angina pectoris. German and Russian authors advise spa therapy. The last section concerns education and deals largely with methods of public dissemination of the suggestions for preventive measures in the third section. Precise labeling of the qualifications of each author would aid in assessing the value of his report. An article recommending exercise from cradle to the grave must be read with the understanding that its proponent is a specialist in physical medicine who knows much about exercise and makes a living prescribing it.

REUBEN BERMAN, M.D.
Minneapolis

Cardiopulmonary Function Tests in Clinical Medicine

by THOMAS P. K. LIM, M.D., *171 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $9.50*

The author keeps his preface promise that ". . . this monograph is written as a primer for those beginners who wish to learn the ABC's of pulmonary and cardiac function tests." As a result, this book should be a useful self-teaching aid for the medical student and physician, whether general practitioner or specialist, who wants to up-date his knowledge of cardiopulmonary function. (However, a proficiency in cardiopulmonary function evaluation probably will not be achieved by the use of this or any book without some associated laboratory or other practical involvement.)

The book and illustrations are of good quality.

The subject matter includes discussions of the applied physiology of ventilation, diffusion and perfusion, and the use of blood gas analyses. A section of illustrative cases is included and there are short sections on phonocardiography and spatial vectorcardiography.

The material is presented in brief, understandable form without sacrificing important details of the principles involved. References to significant medical literature and to newer concepts are given—facilitating the finding of additional information and technical details if needed. Although discussed too briefly considering their probable importance, mention was made of radioactive techniques for estimating ventilation-perfusion relationships. In the section on perfusion the author wisely recognizes the deficiencies which have resulted in part from the artificial separation of the subspecialties of cardiology and pulmonary disease and prefers to deal with the subject as the interdependent system that it is.

I take an opposite view to the author's conclusion that the Van Slyke manometric method remains the method of choice in blood gas analysis. Those physicians who are considering setting up some method for measuring blood gas and pH in their hospital laboratory will find that any available equipment will require professional competence and meticulous care to obtain consistently correct analyses. Van Slyke analyses are advantageous in the cardiac catheterization laboratory for detecting small changes at low blood oxygen levels. However, for most other clinical uses systemic arterial blood is involved, and the modern commercial equipment for measuring pH and partial pressures of oxygen and carbon dioxide directly will likely prove at least as accurate as the Van Slyke apparatus in daily use and is certainly faster. Because of the long plateau of the oxyhemoglobin dissociation curve at the higher ranges of oxygen, clinically significant changes in partial pressure of oxygen in blood (easily detectable with the polarograph) are obscured by very small errors in Van Slyke analysis. Except where abnormalities of hemoglobin might be involved, clinical considerations are best served by measuring, reporting, and thinking about partial pressures of oxygen without conversion to percent oxygen saturation.

This one difference of opinion does not dim in the least my view that this is an excellent book for the physician who wants to use and understand the services of the cardiopulmonary laboratory.

Now if only Dr. Lim or someone as knowledgeable would write a companion book containing all those ". . . minute details of technical and numerical aspects," which he says he purposely (and wisely) left out of this book. Although appealing to a much more limited group of physicians and technologists, such information would be of enormous help in properly equipping and operating new cardiopulmonary laboratories.

JAMES P. LILLEHEI, M.D.
Minneapolis

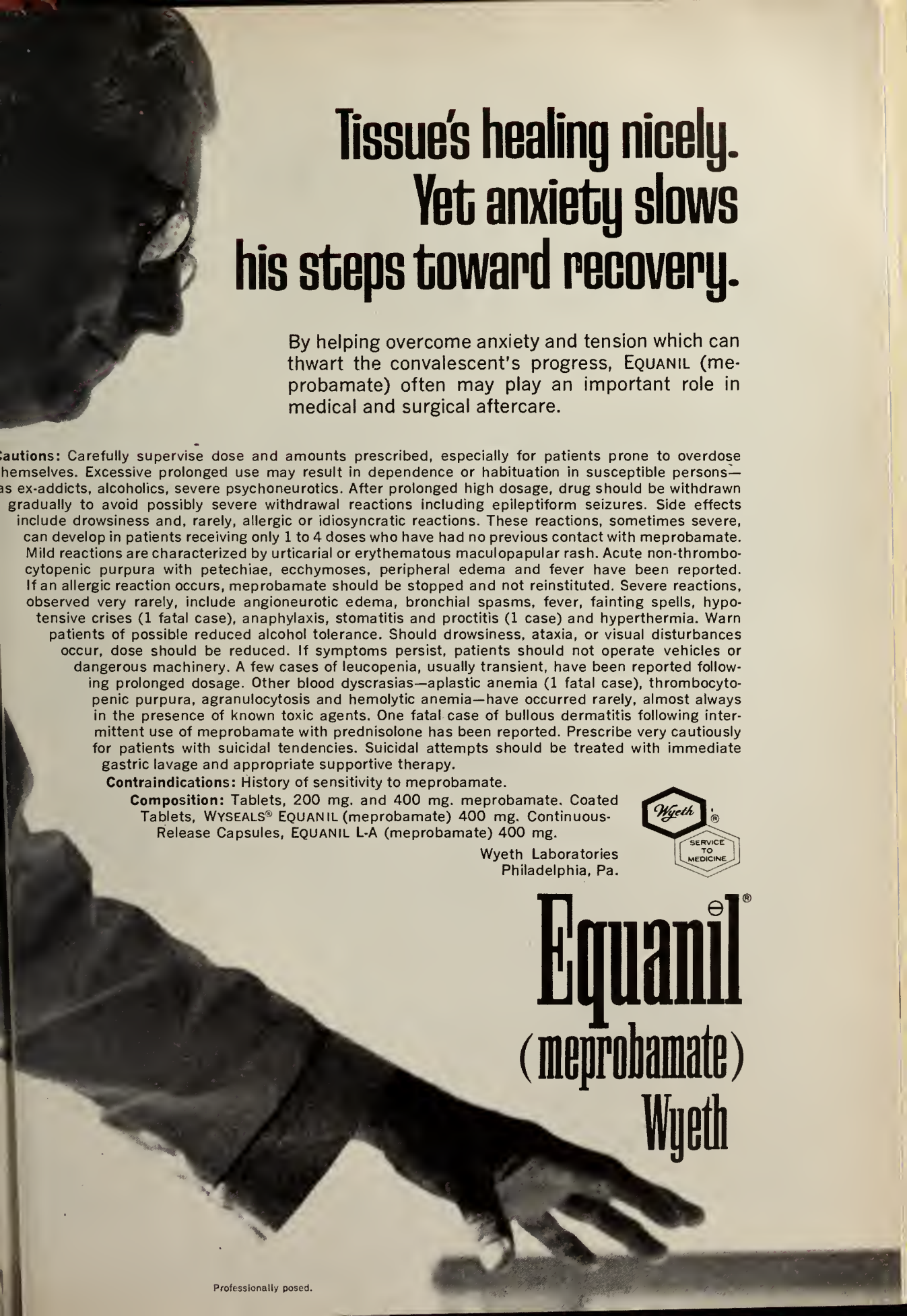
Science and Psychoanalysis, Vol. X: Sexuality of Women

edited by JULES H. MASSERMAN, M.D., *168 pp., Grune & Stratton, Inc., New York, 1966. $7.75*

This small book will interest psychiatrists and psychoanalysts primarily. It contains papers read at the Tenth Annual Meeting of the American Academy of Psychoanalysis and is composed of 2 groups of articles: those

(Continued on Page 14A)





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(Continued from page 11A)

detailing the historical reasons for the split with the American Psychoanalytic Association a decade ago and those dealing with the sexuality of women. This review will confine itself to the latter group.

Of the 9 articles in this category, 7 were written by women. This is somewhat unique since in years gone by, with several notable exceptions, most of the speculations about the formation of femininity were written by men. A number of these articles constitute a critical re-evaluation of the basic Freudian ideas about feminine personality structure. An article by Chodoff reexamined the matter of penis-envy, a concept adanced by Freud which constitutes the foundation of his theories about female personality. As Chodoff points out, if this basic assumption is found wanting, the superstructure built on it falls. He finds no valid evidence to support the theory. Shainess reviewed the various Freudian theories, including the view that the desire for children is a compensation for the lack of a male organ. She found these theories "puzzling, inconsistent, confusing, and potentially absurd."

This volume, then, is of value and interest in that it constitutes a critical look at theoretical positions which, over the past half century, have come to be regarded in some quarters as established fact. The theoretical bedrock, on examination, is found to consist of unsupported opinion and clinical hunch. The book does a service to American psychiatry in calling attention to the fact that theory is theory and must be viewed as such until proved or disproved by the scientific method. Such a position appears to remove most, if not all, Freudian concepts about the development of female sexuality from a factual base. Unfortunately, most such theories do not appear to lend themselves to proof or disproof by the methods of science, for example, the concepts of penis-envy and the Oedipal situation. The moral problems involved in subjecting growing children to scientific manipulation of their psychosexual development appear to be insurmountable.

DONALD W. HASTINGS, M.D.
Minneapolis

Appraisal of Current Concepts in Anesthesiology

edited by JOHN ADRIANI, M.D., *Vol. 3, 522 pp., C. V. Mosby Co., St. Louis, 1966. $10.85*

This is the third volume of a series which originated in 1962 in the anesthesia department of Charity Hospital at New Orleans. It consists of 50 reviews of topics in anesthesiology and allied subjects prepared by staff members and trainees of the department and edited by their director, Dr. Adriani.

The book covers a wide variety of topics of current interest to practitioners of anesthesia and some of which are found in few, if any, other works. For example, Chapter 33 deals with the responsibilities, limits, and liabilities in the testing of new drugs and gives a useful account of what is expected of investigators by the Kefauver-Harris Drug Amendment of the Food, Drug, and Cosmetics Act of 1962, including the subsequent regulations of the Food and Drug Administration. Two other chapters will increase the book's appeal to residents in training. They advise program directors on how to make up examination questions and "what to teach residents in anesthesiology and what they should know."

As with many multiauthored works, this volume suffers from a disconcerting diversity of literary styles and maturities of knowledge. And there are perhaps more than the usual number of errors. These vary from the use of "postnasal" for "postnatal" to the crediting of Louis Pasteur with an observation made by William Pasteur some ten years after the great French chemist's death. But if one overlooks the errors and the few weak chapters he will find here a wealth of material. Each chapter ends with a concise summary and a bibliography usually current through 1965.

The book, which by the way has no illustrations, can be read profitably by all physicians significantly interested in anesthesiology.

JOHN R. GORDON, M.D.
Minneapolis

Congenital Idiopathic Talipes

by JAMES L. LENOIR III, M.D., *354 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $20.50*

This book is the product of many years of research and treatment of congenital Talipes. It is a complete study of the topic including history, etiology, anatomy, kinesiology, pathomechanics, and treatment. Certain sections, such as the history, are very brief but a satisfactory bibliography is provided for those interested in further study. A new comprehensive classification of clubfoot is presented in detail. A significant portion of the book deals with the kinesiology of normal and clubfoot patients and the pathomechanics of the latter. A well illustrated radiographic section is also provided. Operative and cast-on brace type treatment are described, with emphasis on the former. The final chapter consists of illustrations of cases. This book is more suitable as a medical library reference than as a physician's personal text.

H. J. ERICKSON, M.D.
Minneapolis

NEW BOOKS RECEIVED

The Art of Predictive Medicine: The Early Detection of Deteriorative Trends
edited by WEBSTER L. MARXER, M.D., and GEORGE R. COWGILL, Ph.D., 358 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $15.75

Practice of Intramedullary Nailing
by GERHARD KÜNTSCHER, Dr. Med. Habil., 374 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $14

Congenital Idiopathic Talipes
by JAMES L. LeNOIR III, M.D., 354 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $20.50

Essentials of Urography
by ANTHONY F. LALLI, M.D., 150 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $12.75

Shock: Chemistry, Physiology, and Therapy
by WILLIAM C. SHOEMAKER, M.D., 306 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $11.50

Labor: Clinical Evaluation and Management
by EMANUEL A. FRIEDMAN, M.D., 397 pp., ill., Appleton-Century-Crofts, New York, 1967. $13.50

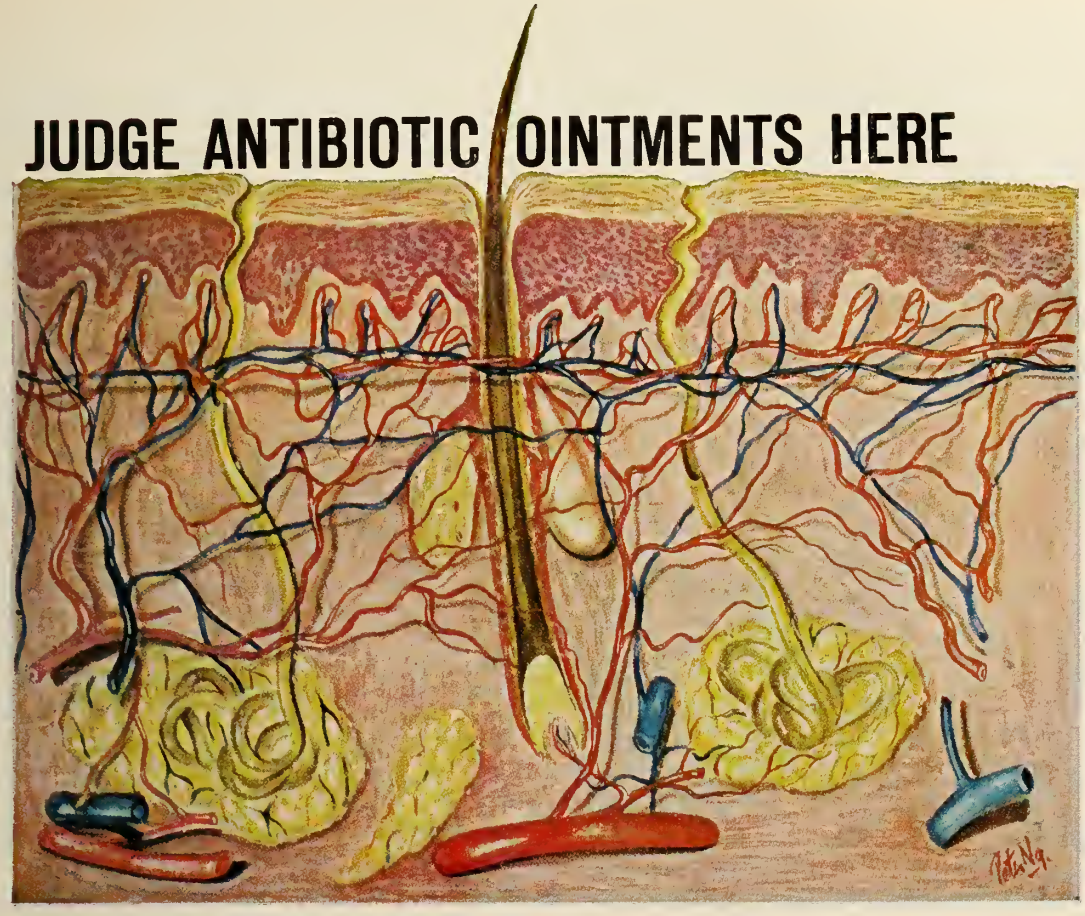
Osler's Textbook Revisited
edited by A. McGEHEE HARVEY and VICTOR A. McKUSICK, 361 pp., ill., Appleton-Century-Crofts, New York, 1967. $6.75

Cardiac Surgery
edited by JOHN C. NORMAN, M.D., 603 pp., ill., Appleton-Century-Crofts, New York, 1967. $15 (cloth), $9.75 (paperback)

Teaching and Display Techniques in Anatomy and Zoology
by T. F. SPENCE, B.Sc., 181 pp., ill., Pergamon Press, New York, 1967. $5

(Continued on Page 16A)

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One Hundred Years of Medical Progress: A History of the New York Medical College Flower and Fifth Avenue Hospitals
by LEONARD PAUL WERSHUB, M.D., 259 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $6.50

International Encyclopedia of Pharmacology and Therapeutics, Section 6, Volume 1: Clinical Pharmacology
edited by L. LASAGNA, 373 pp., ill., Pergamon Press, Long Island City, 1966. $12.50

International Encyclopedia of Pharmacology and Therapeutics, Section 6, Volume II: Clinical Pharmacology
edited by L. LASAGNA, 667 pp., ill., Pergamon Press, Long Island City, 1966. $9.50

Neurological and Neurosurgical Nursing: An Introduction
by DONALD F. SCOTT, M.B., and BARBARA DODD, S.R.N., 204 pp., ill., Pergamon Press, Long Island City, 1966. $6

Radiology in World War II
edited by KENNETH D. A. ALLEN, M.D., 1132 pp., ill., U.S. Government Printing Office, Washington, 1966. $8.25

Psychopathic Disorders and Their Assessment
edited by MICHAEL CRAFT, M.D., 320 pp., ill., Pergamon Press, Long Island City, 1966. $8

Amines and Schizophrenia
edited by HAROLD E. HIMWICH, SEYMOUR S. KETY, and JOHN R. SMYTHIES, 290 pp., ill., Pergamon Press, New York, 1967. $11.50

Elementary Statistics: A Workbook
by Dr. K. Hope, 101 pp., ill., Pergamon Press, New York, 1967. $4

Present-day Russian Psychology: A Symposium by Seven Authors
edited by NEIL O'CONNOR, Ph.D., 201 pp., ill., Pergamon Press, New York, 1966. $6.50

Congestive Heart Failure
by RALPH M. MYERSON, M.D., and BERNARD H. PASTOR, M.D., 174 pp., ill., C. V. Mosby Co., St. Louis, Mo., 1967. $12.85

Vascular Complications of Diabetes Mellitus: With Special Emphasis on Microangiopathy of the Eye
edited by SAMUEL J. KIMURA, M.D., and WAYNE M. CAYGILL, M.D., 277 pp., ill., C. V. Mosby Co., St. Louis, Mo., 1967. $19.75

Trachoma: Prevention and Treatment
by GIAMBATTISTA BIETTI, M.D., and GEORGES H. WERNER, Ph.D., 227 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $11.50

Progress in Brain Research: Volume 20: Pharmacology and Physiology of the Reticular Formation
edited by A. V. VALDMAN, 339 pp., ill., Elsevier Publishing Co., New York City, 1967. $21.75

Biochemical Factors in Alcoholism
edited by ROGER P. MAICKEL, 254 pp., ill., Pergamon Press, New York City, 1967. $12

Ciba Foundation Symposium: Ethics in Medical Progress
edited by G. E. W. WOLSTENHOLME, F.R.C.P., and MAEVE O'CONNOR, 257 pp., Little, Brown & Co., Boston, 1966. $11.75

Ciba Foundation Symposium: Principles of Biomolecular Organization
edited by G. E. W. WOLSTENHOLME, F.R.C.P., and MAEVE O'CONNOR, 491 pp., ill., Little, Brown & Co., Boston, 1966. $15

Laboratory Tests in Common Use
by SOLOMON GARB, M.D., 4th ed., 192 pp., Springer Publishing Co., Inc., New York City, 1966. $2.85 (paperback)

Differential Diagnosis: A Memory Stimulus System for Pocket, Punchcard and Computer
by STANLEY B. BERKOWITZ, M.D., 333 pp., Charles C Thomas Co., Springfield, Ill., 1967. $11.50

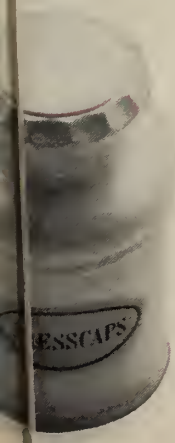
Early Childhood Autism: Clinical, Educational and Social Aspects
edited by J. K. WING, 333 pp., ill., Pergamon Press, New York City, 1966. $7.50

(Continued on page 18A)



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NEW BOOKS RECEIVED

(Continued from Page 16A)

A Short Textbook of Medicine
by J. C. HOUSTON, M.D., C. L. JOINER, M.D., and J. B. TROUNCE, M.D., 2d ed., 573 pp., J. B. Lippincott Co., Philadelphia, 1966. $7.50 (paperback)

Smithsonian Year 1966: Annual Report of the Smithsonian Institution, for the year ended June 30, 1966, including the Financial Report of the Executive Committee of the Board of Regents
409 pp., U.S. Government Printing Office, Washington, D.C. $1.75

The Research Process in Nursing
by DAVID J. FOX, Ph.D., and RUTH LUND KELLY, R.N., 611 pp., Appleton-Century-Crofts, New York, 1967. $12.95

ANA Clinical Sessions: American Nurses' Association
1966, San Francisco, 272 pp., Appleton-Century-Crofts, New York, 1967. $8.50

Choice in Human Affairs:
An Application to Aging-Accident-Illness Problems
by ELMER LUCHTERHAND and DANIEL SYDIAHA, 176 pp., ill., College & University Press, New Haven, Conn., 1966. $5

Medical Terminology for the Nonprofessional Employee
by MARY LYLE, 186 pp., 1st ed., Mail Box, Walnut Creek, Calif., 1967.

Ward Procedures and Techniques
by PHILIP COOPER, M.D., 303 pp., ill., Appleton-Century-Crofts, New York, 1967. (Price requested)

The Assessment of Nutritional Status of the Community
by DERRICK B. JELLIFFE, M.D., 271 pp., ill., World Health Organization, Geneva, 1966. $6

Scoliosis: A Manual of Concept and Treatment
by STANLEY HOPPENFELD, 115 pp., ill., J. B. Lippincott Co., Philadelphia, 1967. $7

Pneumoencephalography
by E. GRAEME ROBERTSON, M.D., 2d. ed., 808 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $30.50

Sensory Mechanisms (Progress in Brain Research, 23)
edited by Y. ZOTTERMAN, 225 pp., ill., Elsevier Publishing Co., New York, 1967. $14.50

Pathology Annual: Volume 2, 1967
series editor, SHELDON C. SOMMERS, M.D., 427 pp., ill., Appleton-Century-Crofts, New York City, 1967. $13.75

Pulmonary Insufficiency and Respiratory Failure
by GILES F. FILLEY, M.D., 162 pp., ill., Lea & Febiger, Philadelphia, 1967. $7

Shadow and Substance: The Story of Medical Radiography
by PINO DONIZETTI, 213 pp., ill., Pergamon Press, New York City, 1967. $6

Bronchial Asthma:
A Genetic, Population and Psychiatric Study
by DENIS LEIGH, M.D., and EDWARD MARLEY, M.D., 192 pp., Pergamon Press, New York City, 1967. $10

The Acute Abdomen and Emergent Lesions of the Gastrointestinal Tract
compiled and edited by HERBERT R. HAWTHORNE, M.D., ALFRED S. FROBESE, M.D., and JULIAN A. STERLING, M.D., 485 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $29.50

The Chemistry of the Gonadotrophins
by WILFRED R. BUTT, Ph.D., 116 pp., Charles C Thomas Co., Springfield, Ill., 1967. $7.50

Surgery for Acquired Coronary Disease
by WILLIAM H. SEWELL, M.D., 377 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $18.50

Support of the Failing Circulation:
The Use of a Pump Oxygenator in Clinical Cardiac Failure
by JOHN HINES KENNEDY, M.D., 138 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $7.50

German-English/English-German: A Dictionary of Professional Terminology of Speech Pathology and Audiology
by PETER B. MUELLER, 53 pp., Charles C Thomas Co., Springfield, Ill., 1967. $5

News Briefs . . .

North Dakota

OFFICERS ELECTED at the annual meeting of the North Dakota Physicians Service (Blue Shield) were: Dr. H. A. Norum of Fargo, president; Dr. W. B. Huntley of Minot, vice-president; Dr. A. R. Gilsdorf of Dickinson, secretary; Dr. W. T. Powers of Grand Forks, treasurer; and Dr. F. A. DeCesare of Fargo, renamed delegate to national and district Blue Shield meetings.

* * * *

ELECTED at the annual convention of the North Dakota Medical Association were: Dr. V. J. Fischer of Minot, president; Dr. C. H. Peters of Bismarck, president-elect; Dr. Robert Painter of Grand Forks, first vice-president; and Dr. T. E. Pederson of Jamestown, second vice-president. Reelected were: Dr. P. O. Dahl of Bismarck, speaker of the house of delegates; Dr. W. M. Buckingham of Bismarck, secretary; and Dr. R. D. Nierling of Jamestown, treasurer. Fifty-year pins were presented to Dr. Wilbur L. Diven of Bismarck and Dr. G. Arthur Larson of Fargo.

* * * *

DR. JON V. EYLANDS of Rugby worked for a month on his main interest, prostatic surgery, at Whippscross Hospital and the Leicester Royal Infirmary, London.

* * * *

DR. WALLIS L. CRADDOCK, formerly of Palo Alto, Calif., was named chief of staff for the Fargo Veterans Administration Hospital, succeeding Dr. Wallace Pianka.

* * * *

DR. ROBERT A. LIM, formerly of Chicago, was appointed staff anesthesiologist at St. Alexius Hospital, Bismarck.

* * * *

DR. WALTER GOVAKI, a native of India, began practice in Mohall. The community now has three physicians.

Minnesota

DR. OWEN H. WANGENSTEEN, chairman of the department of surgery at the University of Minnesota for thirty-seven years, was honored by three days of activities before his June 30 retirement. Two of the days were scientific sessions at which 47 papers were presented.

* * * *

DR. JOHN L. JUERGENS of the Mayo Clinic, Rochester, was elected president of the Minnesota Heart Association at its annual meeting.

* * * *

DR. JAMES TERRIAN, formerly of Thief River Falls, will join the Klefstad Clinic at Greenbush in July.

* * * *

DR. L. J. OPSAHL of Willmar was appointed coroner of Kandiyohi county.

* * * *

DR. CHARLES W. MAYO of Rochester resigned as chairman of the board of regents of the University of Minnesota. He said he would continue as a regent until his term expires in February 1971.

(Continued on Page 20A)

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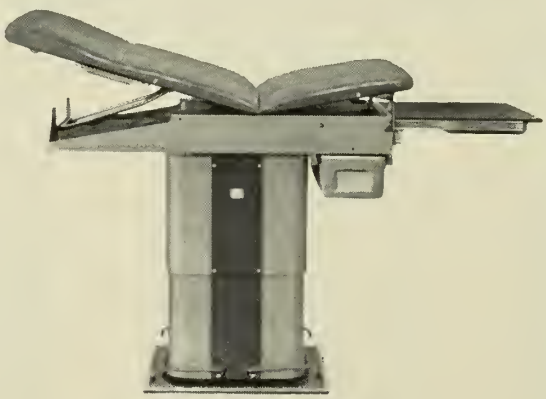
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NEWS BRIEFS

(Continued from Page 19A)

DRS. FRANCIS M. MCCARTEN, RUSSEL E. CARLSON, and EDWARD B. KIOLBASA of Stillwater held an open house at their newly completed offices.

* * * *

DR. ROBERT L. MERRICK of North Oaks was elected president of the medical staff of Miller Hospital, St. Paul.

* * * *

DRS. GAIL HAUT, JOHN FINCH, and JOSEPH HEANEY resigned from the Mankato Clinic to take positions elsewhere.

* * * *

DR. WILLIAM S. POLLARD of Duluth was elected to membership in the Harvey Cushing Society at its annual meeting in San Francisco.

* * * *

DR. FRANK ROBERT ARKO, JR., of Hibbing was elected to active membership in the American Academy of General Practice.

* * * *

DR. JOHN N. KING, a resident in surgery at the Mayo Graduate School of Medicine, volunteered for two months duty in Vietnam as part of a project sponsored by the American Medical Association.

* * * *

DRS. ABDUR and JILL RAHMAAN, who have joined the Buffalo Lake Clinic, were honored at an open house.

* * * *

THE FEDERAL GOVERNMENT granted $506,000 for a diabetes detection and education center which will be located in the east wing of Asbury Methodist Hospital, Minneapolis.

* * * *

DRS. CLAIRE STROBEL and HOWARD SNIDER of the Mankato Clinic have made their surgical services available to the Wells Municipal Hospital.

* * * *

HONORED by Gov. Harold LeVander's Commission on Employment of the Handicapped were Dr. Earl Elkins, senior consultant in Mayo Clinic's section of physical medicine and rehabilitation, who was named Minnesota Physician of the Year; and Dr. Christian Zaun of St. Paul, who was cited for his work in helping the handicapped return to a productive place in society.

* * * *

NOTED PHYSICIANS invited to the 114th annual meeting of the Minnesota State Medical Association at Duluth included Dr. Alan F. Guttmacher, president of the Planned Parenthood Federation of America, New York, and Dr. F. J. L. Blasingame, executive vice-president of the American Medical Association.

* * * *

DR. JOHN HILL of the Mayo Clinic was named president of the Rochester Board of Public Health.

* * * *

DR. JOHN WILTSIE was appointed a consultant in internal medicine at the Mayo Clinic.

* * * *

DR. GORDON O. GREENE of Arlington left his partnership in the Arlington Clinic to enter a three-year specialty program of study at the Mental Health Institute, Cherokee, Iowa.

* * * *

DR. JOHN C. MICKELSON terminated his twenty-eight-year association with the Mankato Clinic to practice urology in Los Angeles.

NEWS BRIEFS

MR. THOMAS BERGSTROM, a medical student at Bowman-Gray School of Medicine, Winston-Salem, N.C., will train for six weeks, starting in July, with Dr. James Kvale at the Atwater Community Medical Center.

* * * *

DR. GARY R. FEIGAL of Oxnard, Calif., will join Drs. E. C. Bayley and D. W. Sontag in practice in Lake City, starting in October.

* * * *

DR. ROBERT A. VAN TASSEL of Watertown is serving a three-month temporary tour of duty as a flight surgeon at Mildenhall RAF Station, England.

* * * *

ADDITIONS to the staff of Methodist Hospital, St. Louis Park, are Drs. Thomas H. Comfort, orthopedist; Roger I. Gruys, surgeon; Evan L. Nelson, otolaryngologist; Alvin Shemesh and Robert D. Thomasson, general practitioners; and James F. Reynolds, internist.

* * * *

DR. H. A. MOLANDER of Maplewood was honored by the Minnesota State Medical Association upon completing fifty years in medical practice.

* * * *

DR. JAMES C. CAIN of the Mayo Clinic, Rochester, was appointed by President Johnson as one of the 3 U.S. delegates to the 20th World Health Assembly of the World Health Organization held in Geneva, Switzerland.

* * * *

SPECIAL CITATIONS were presented at the Minnesota State Medical Association annual banquet to 3 University of Minnesota professors: Dr. Owen C. Wangensteen, retiring chairman of the department of surgery; Dr. C. J. Watson, former chairman of the department of internal medicine; and Dr. J. A. Myers, former professor of medicine in preventive medicine and public health.

South Dakota

DR. D. MAX READE of the Yankton Medical Clinic attended a meeting of leading cardiologists in Minneapolis.

* * * *

WATERTOWN was host to the 15th annual meeting of the South Dakota Heart Association.

Deaths . . .

DR. THOMAS J. DEVEREAUX, 76, Wayzata, Minn., died April 30. He was to be honored May 16 by the Minnesota Medical Association for fifty years of service in the medical profession.

* * * *

DR. JOHN G. W. HAVENS, 90, formerly of Austin, Minn., died at Minneapolis, May 6. He had practiced medicine at Austin for forty-four years, retiring one month before his 87th birthday.

* * * *

DR. J. WILBUR MORELAND, 91, formerly of Carpio, N.D., died at Minot, April 23. He had been honored by the North Dakota Medical Association in 1963 for completing fifty years of practice in the state.

* * * *

DR. BENJAMIN I. DERAUF, 73, Roseville, Minn., died May 21. He had retired in 1962 after forty-three years as chief surgeon at Northern Pacific Hospital, St. Paul.

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GENERAL PRACTITIONER wanted—to join established practitioner. Lacoted 65 miles north of Minneapolis-St. Paul oreo. Papulation over 3,000. Hospitol in tawn. Gaad schaals, churches, and fine fishing ond recreatianal area. Contact A. C. Petersan, M.D., 200 E. Forest, Mora, Minnesota 55051.

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1 Sherman, L. F.: Minnesota Med. 48,605. May, 1965. 2 Sherman, L. F. Personal Communication.

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CORTENEMA consists of a suspension of partially solubilized hydrocortisone alcohol in an aqueous vehicle. Thus, it provides the potent anti-inflammatory effect characteristic of the corticosteroids. Because hydrocortisone alcohol is absorbed to some extent from the colon, CORTENEMA provides both topical and systemic actions.

Indications: CORTENEMA Hydrocortisone Retention Enema is indicated as an adjunct in the treatment of idiopathic nonspecific ulcerative colitis. It is most useful in cases involving the rectum, sigmoid and left colon, where it exerts a topical effect. Due to its systemic action, CORTENEMA has proved effective in certain cases extending to the transverse and ascending colons.

Rectal steroid therapy brings about prompt symptomatic and proctologic remissions in a significant proportion of patients, but it apparently does not alter the recurrent and progressive nature of ulcerative colitis. Therefore, this form of treatment is not recommended for prophylaxis during periods of remission.

Contraindications: Local contraindications to the use of intrarectal steroids include obstruction, abscess, perforation, peritonitis, fresh intestinal anastomoses, extensive fistulas and sinus tracts. Tuberculosis (active, latent or questionably healed), ocular herpes simplex, and acute psychosis are usually considered absolute contraindications to the use of corticosteroids.

Relative contraindications include active peptic ulcer, acute glomerulonephritis, myasthenia gravis, osteoporosis, diverticulitis, thrombophlebitis, psychic disturbances, pregnancy, diabetes, hyperthyroidism, acute coronary disease, hypertension, limited cardiac reserve, and local or systemic infections, including fungal or exanthematous diseases. Where these conditions exist, the expected benefits from CORTENEMA must be weighed against the risks involved in its use.

If clinical or proctologic improvement does not occur within two or three weeks after starting CORTENEMA, discontinue its use.

Precautions: CORTENEMA Hydrocortisone Retention Enema should be administered with caution in patients with severe ulcerative disease because these patients are predisposed to perforation of the bowel wall. In the advanced stages of chronic ulcerative colitis, where there is loss of mucosa, and thickening and fibrosis of the bowel wall, steroid therapy theoretically might hasten deterioration, although this has not been proved with steroids in actual practice.

In severe cases, where surgery is imminent in the absence of marked clinical improvement, it is hazardous to wait more than a few days for a satisfactory response to medical treatment.

Because of the possibility of adrenal insufficiency caused by suppression of the adrenal cortex by glucocorticoids especially after prolonged therapy, it is important that therapy be withdrawn gradually. If the patient is subjected to unusual stress, while on therapy or up to a year after discontinuation of steroids, adequate supportive measures and increased or reinstated systemic steroid therapy are indicated. In the case of surgery, these measures should be continued throughout the pre- and postoperative recovery periods, bearing in mind the possible deleterious effects of corticosteroids on fresh intestinal anastomoses.

Steroid therapy might impair prognosis in surgery by increasing the hazard of infection. If infection is suspected, appropriate antibiotic therapy must be administered, usually in larger than ordinary doses.

General precautions common to all corticosteroid therapy should be observed during treatment with CORTENEMA, including those pertaining to growth suppression in children during prolonged use. Patients should be kept under close observation. If severe reactions or idiosyncrasies occur, steroids should be discontinued immediately and appropriate measures instituted.

If it is necessary to use CORTENEMA in pregnant patients, their infants should be closely observed for signs of hypoadrenalism; and appropriate measures, including administration of corticosteroids, should be instituted if such signs are seen.

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Adverse Reactions: CORTENEMA Hydrocortisone Retention Enema may produce all of the side effects known to occur with other forms of hydrocortisone therapy. These include moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use of all corticosteroids results in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of the hip and humerus may occur as part of this catabolic phenomenon.

Where hypopotassemia and the other symptoms associated with fluid and electrolyte imbalance call for potassium supplementation and salt-poor or salt-free diets, these may be instituted and are compatible with the diet requirements for ulcerative colitis.

Dosage and Administration: The use of CORTENEMA Hydrocortisone Retention Enema as an adjunct in the treatment of ulcerative colitis is predicated upon the concomitant use of modern supportive measures, such as blood replacement, rational dietary control, antispasmodics and sedatives, antibacterial therapy, and systemic steroids.

The usual dose is one CORTENEMA Hydrocortisone Retention Enema daily for two or three weeks and every second day thereafter, administered intrarectally in the evening before retiring. Every effort should be made to retain the medication for at least one hour, preferably all night. This may be facilitated by prior sedation and or antidiarrheal medication. If clinical or proctologic improvement fail to occur within two or three weeks, discontinue the drug.

For administration by retention enema, instruct the patient to lie on his left side during instillation. Shake the bottle vigorously to resuspend the insoluble portion of hydrocortisone. Expose the lubricated tip by removal of the protective sheath, grasping the bottle at the neck where it is most rigid. Carefully insert the lubricated tip into the rectum in the direction of the sacrum. Slowly express the contents by compressing the container. After instillation, the patient should remain on his left side for at least 30 minutes, to allow distribution of the medication throughout the descending colon and rectum.

For coverage of more distant segments of the colon, CORTENEMA Hydrocortisone Retention Enema may be diluted to from 100 cc. to 300 cc. with isotonic saline and administered by intrarectal drip.

The duration of treatment is dependent on the degree of response. If a satisfactory response is to be obtained, it usually occurs within five to seven days, as evidenced by a marked reduction of clinical symptoms. Improvement in the appearance of the mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. The usual duration of therapy is two weeks, although some cases require therapy for longer periods (up to two or three months). Minimal control of symptoms is an insufficient basis for the prolonged use of CORTENEMA. If there is no clinical or proctologic response within two or three weeks, or if the patient's condition worsens, discontinue the drug.

Because of hydrocortisone absorption, proper precautions against unwanted systemic reactions or side effects should be observed. Symptomatic improvement, evidenced by decreased diarrhea, weight gain, improved appetite, lessened fever, and decrease in leukocytosis, may be misleading and should not be used as the sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, enema or drip therapy should not be employed in the immediate or early postoperative period following ileorectostomy.

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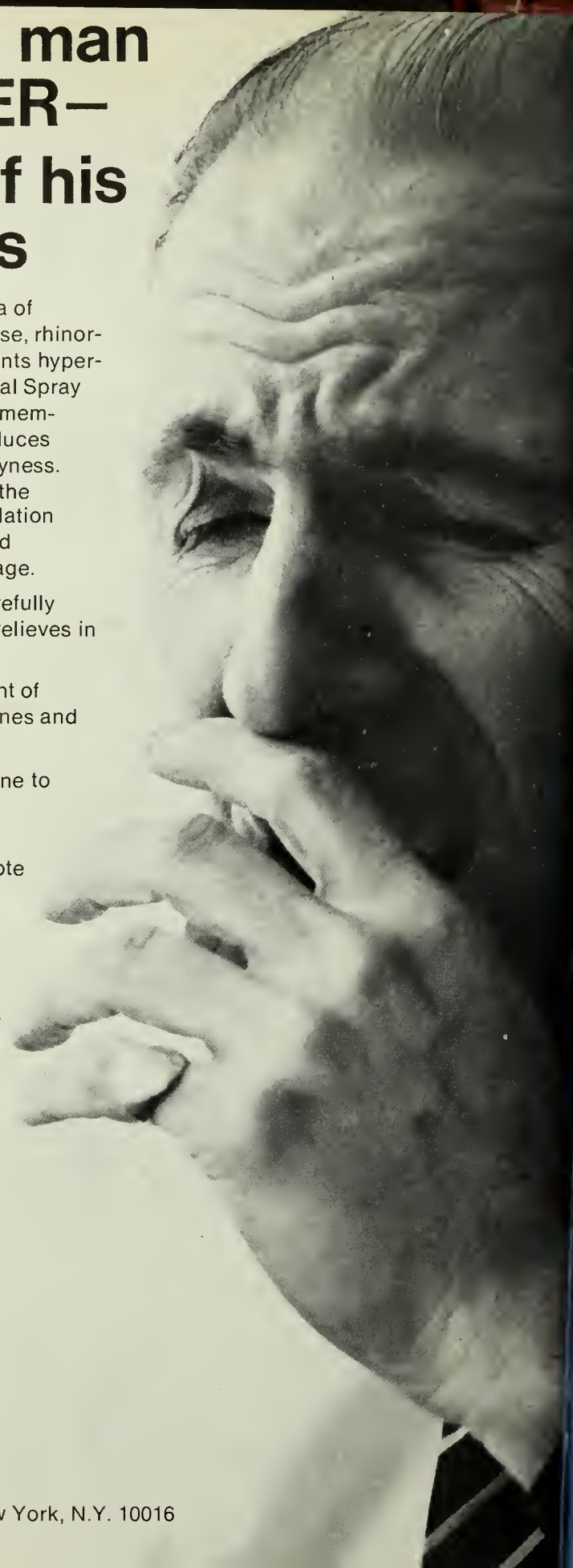
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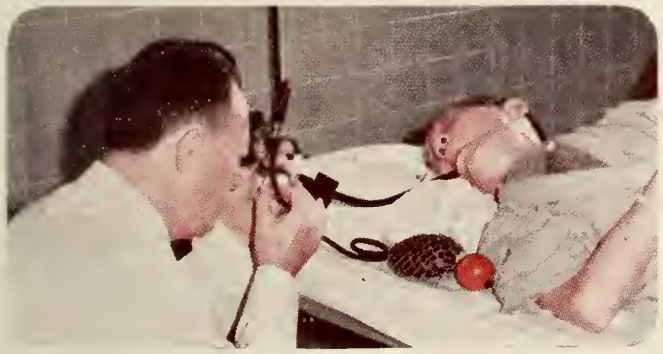
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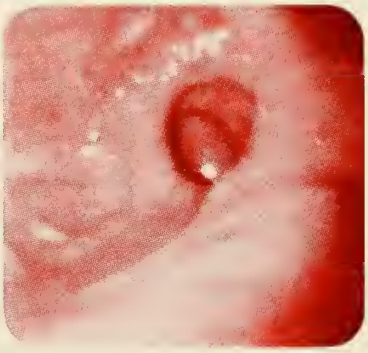
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1. Barowsky, H.; Greene, L.; Bennett, R., and Buganza, G.: The Effect of Anticholinergic Drugs on Gastric Motility and Pyloric Function, Scientific Exhibit, Annual Convention of the American Medical Association, Chicago, Illinois, June 26-30, 1966.

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CONTENTS

Incompetency 273
James K. O'Toole, M.D.

Circadian Rhythms and the Pineal Gland 277
R. E. Wiederanders, M.D., K. A. Lind, and G. W. Evans, B.S.

Acute Bronchiolitis as a Cause of Chronic Obstructive Lung Disease in Adults: Report of Two Cases 281
David E. Dines, M.D.

Section on Pain and Distress:
The Mystery of Atypical Pain 283
John S. Lundy, M.D.

Cerebral Vasospasm in Migraine 283
Donald J. Dalessio, M.D.

Control of Pain in Patients with Cancer 287
Guy Owens, M.D.

Dr. Ralph Moore Tovell 291
John S. Lundy, M.D.

Book Reviews on Pain 292

Pregnancy in Patients with Osteogenesis Imperfecta: A Case Report and a Review of the Literature 293
Charles Dunham, B.S., and William Spellacy, M.D.

Transactions of the North Dakota State Medical Association 296

Book Reviews 13A

News Briefs 20A

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IN BRIEF.

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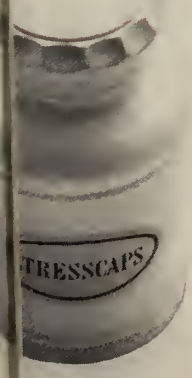
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SERVES THE MEDICAL PROFESSION OF THE UPPER MIDWEST

Incompetency

JAMES K. O'TOOLE, M.D.

Bismarck

MENTAL OR EMOTIONAL illness is one of the great concerns in our country today. More than half of the hospital beds are occupied by individuals suffering from some emotional illness. One in 10 persons will need treatment for mental illness some time in his life, and this will affect 1 out of every 3 families in the United States.

Medicolegal problems affecting the mentally ill are challenging, and they point up the need for further study and research. Each year, a quarter of a million people are committed to mental institutions. Three times that number are sentenced to state and federal prisons annually. Most of these people have some type of emotional difficulty.

The law not only determines who should be committed and by what procedure but it also resolves many related questions. For instance: Should a person committed to a mental hospital or prison be declared incompetent? May a mental patient be sterilized or given other extreme medical treatment? Should a mental patient be allowed to see his lawyer or the clergyman whenever he chooses, even though it disrupts his treatment program? Should he be allowed to vote? Can he sue or be sued? When, and under what conditions, should the committed person be discharged?

JAMES K. O'TOOLE *is psychiatric consultant to the Public Welfare Board of North Dakota, Bismarck.*

The interest of the law also extends into many other facets pertaining to the mentally ill person who is not under custodial care. It is a legal function to determine if the mentally ill are to be declared incompetent and placed under guardianship; to determine whether or not the mentally ill are to be deprived of their civil rights; and to determine whether or not the mentally ill shall be allowed to marry, divorce, enter into a contract, or prepare a will. It is also under the court's domain to decide whether or not the mentally ill person should be liable for taxes or responsible for damages to others. It is a legal function to determine how insanity or mental illness is defined in criminal and civil cases. There is hardly a facet of the law in which the particular status or the interests of the mentally ill do not pose special problems.

I would like to discuss the topic of incompetency and its medicolegal implications. Incompetency is an ambiguous word with varying interpretations, and it involves an area in which law and medicine overlap. Many physicians prefer to avoid this area not only because of the psychologic trauma to the physician created by testifying in court but because the many interpretations of incompetency lend to confusion. How is incompetency determined? Can anyone really know when another person is incompetent? Can physicians really tell the court that a person is competent or incompetent? In the matter of probate (estates and wills), the physician is often called to give an opinion on

testimony about some individual. Often, he does not have a chance to examine this person. Often, courts ask the physician to give an opinion about a person who has done something in the past, or the court may ask if a person may become incompetent in the future. Thus, the physician is expected to be an oracle in forecasting the future, and, realizing the multiple causality of actions, the physician prefers to avoid this type of testimony.

Although this point may not yield to statistics, it is my belief that, before death comes, every man or woman is liable to be temporarily or permanently unable to manage his business affairs. Heart attacks, cerebral hemorrhages, ordinary illnesses, accidents, senility, or emotional problems may be some of the causes.

LEGAL DEFINITIONS

North Dakota law has specifically avoided defining the term "incompetency," but the term is used occasionally in the Century Code (29-20-02). Incompetency to stand trial in North Dakota is defined as a person who is " . . . unable to understand the proceedings against him or to assist in his defense. . . ."

While using the term "incompetency," the code (14-01-03) also uses the synonym "incapacity" when it states that an insane person cannot contract: "After his incapacity has been determined judicially upon application for the appointment of a guardian, a person of unsound mind can make no conveyance or other contract, nor delegate any power, nor waive any right until his restoration to capacity is determined judicially."

Thus, it seems that appointment of a guardian is tantamount to the declaration of incompetency or incapacity, for guardianship is closely related to incompetency. The North Dakota constitution (Article 4, Section III) gives "exclusive original jurisdiction . . ." to the county court in the appointment of guardians and administrators. Section 30-10-02 of the North Dakota Century Code reiterates the constitution and authorizes the county courts to appoint ". . . a guardian, if jurisdictional fact exist, of the person or estate, or both, of a person residing in this state who is: [1] a minor who has no guardian legally appointed by will or deed, [2] an habitual drunkard who is incompetent to manage his own property, [3] a spendthrift, or [4] a person who is of unsound mind, or from any cause mentally or otherwise incompetent to manage his own property."

Section 30-10-05 describes the necessary petition for appointment of guardian. It states: "The appointment of a guardian for a minor, habitual drunkard, spendthrift, or incompetent person must be made by a county court, and letters of guardianship must issue if: [1] a petition of a relative or other person praying for such appointment is filed in county court, [2] such petition shows: the jurisdictional facts, the circumstances making appointment of a guardian necessary or convenient, and the description and value of the property owned by the minor, habitual drunkard, spendthrift, or incompetent, if any, and the probable income therefrom, [3] a citation has been served . . . and [4] a hearing has been held upon such petition, and [5] it is found by the court that the appointment prayed for is necessary or convenient."

Section 30-10-20 describes the procedure of restoration of competency through a county court hearing, and it implies that one is competent who is ". . . capable of taking care of himself and his property."

It has been evident from reports that many physicians and lawyers believe commitment to the state hospital is the same as declaration of incompetency and necessitates the appointment of a guardian. This impression is completely erroneous. North Dakota Law 25-03-20 preserves all civil rights of these hospitalized patients unless adjudicated incompetent by the court. These patients volunteer or are committed to the state hospital for treatment of some mental disorder. They are not confined because of incompetency. A seriously ill person may be both committed and incompetent. This is the exception, not the rule. Commitment to the state hospital is performed by the county mental health board, whereas the county court holds a hearing and has a formal procedure to accomplish adjudication of incompetency and to appoint a guardian.

DISCUSSION

Determination of incompetency or appointment of a guardian for a person of unsound mind means that, because of such an illness, this person cannot manage his own business affairs or property or otherwise cannot cope with ordinary affairs of life and needs someone to handle these matters for him to protect his interests.

As stated before, Section 30-10-05 of the North Dakota Century Code describes the necessary petition for appointment of a guardian and, among others things, allows for a hearing upon such petition. The law requires that such petition show circumstances necessitating the appointment of a guardian. Unless incompetency, necessity, or convenience is clearly demonstrat-

ed, a person is presumed to be able to manage his own affairs. Thus, the burden of proof is on the person who would declare another person incompetent. The petition must show positive and objective proof that the person is, indeed, incompetent. The petition must show that a person has, in fact, acted in a manner which would suggest incompetency, that is, squandering money, being unable to handle his person, or appearing malnourished or in a "filthy state."

Generally, judges require a three-step process at the hearing to determine mental incompetency. If any of these steps are not proved, then the subject remains competent.[1]

1] It must be shown that the subject has a mental disorder. This is often where the physician enters the case. Unfortunately, this requires placing a pejorative label on a person. Too often, this results in identifying a person in terms of a disability, even though the disability or condition may be of short duration, of a minor degree, or not too different from many others not so labeled. In a sense, it is calling a person a bad name, using such terms as psychosis, schizophrenia, neurotic, mental retardate, and so forth. Even though we have difficulty in labeling, we do realize that there are some people who cannot carry out their businesses, at least for a short time, and some type of adjudication is necessary.

2] The mental disorder must be shown to cause some impairment of judgment. This is often as difficult as showing that a person has a mental illness. A psychiatrist may be able to demonstrate that a person is depressed, has hallucinations, or has severe delusions, but this is not enough. Specific instances of poor judgment must be evinced by the psychiatrist or the other witnesses. It must be shown how the patient squandered money or how he used poor judgment in driving, in marrying, or in caring for himself.

3] It must be shown that the defect in judgment, as a practical matter, causes a specific incapacity with reference to the matter in question, such as driving a car, voting, marrying, divorcing, contracting, or managing business dealings. This is related to the second procedure but is more specific. On this point, the physician should be prepared to answer the following questions: Will the person squander money imprudently because of the mental disorder? Is the person likely to fall prey to designing persons because of gullibility or suggestibility, and, if so, is this gullibility or suggestibility due to the mental disorder or is it similar to the poor judgment that any normal person might have? Is the person likely to hoard money and allow his family to do without necessities because of an unreasonable fear of poverty or because of some delusion which will make it impossible for him to negotiate checks?

A particular area of incompetency is the testamentary capacity to make a will. In this case, the court has a few different qualifications, and since the person has died, the physician has an even more difficult task—he cannot examine the person.

To invalidate a will is more difficult than to void a contract or to have an individual declared mentally incapacitated. The wording of a will must objectively show that, at the time of the writing of the will, this person lacks testamentary power, lacks testamentary capacity, or that he may have presence of undue influence.[2]

The laws pertaining to wills have been maintained with few modifications by some states since 1870, when Lord Chief Justice Cockburn of England pointed out the three major areas that must suggest incapacity before a will is declared void: [1] The person does not know that he is making a will—that this document is made to dispose of his properties after his death. [2] The person does not know the nature and extent of his properties, or he has delusions of grandeur, riches, or even poverty. [3] The person must know the natural recipients of his bounty; that is, he must know his heirs generally by name so that, in a sense, he can know to whom the property would go if he were to die without a will. We frequently hear of wills that list the names of every person to whom the testator may be related. The testator uses this as a safeguard against anyone who would later question his testamentary capacity. Lawyers tell me that this is not necessary and is only done as a protection against people who might contest the will.

One facet of our laws that needs more consideration and investigation is a simplified method for establishing voluntary guardianship in cases of physical incapacity, such as when a patient may sustain a severe heart attack, a stroke, or other incapacitating, long-term physical disability. The scope of psychologic, physical, and economic damage to this family and the patient is immeasurable if the patient is flat on his back, recovering from some severe illness, and yet required to carry on his normal business.

Voluntary psychologic incapacity, apparently, has been partially tested in North Dakota in the case of the guardianship of Jones (66 N.D. 185, 263 NW 160, 120 ALR 441), where a superior

court ruled that: "Where a person of lawful age personally appears without being cited at a hearing called by a county judge on petition for a guardian's appointment, and states that she wishes to have a person appointed as guardian, and signs a written request for his appointment, the court acquires jurisdiction over her person to the same extent as if she had been cited."

Thus, it would seem that, although not specifically tested in North Dakota, we do have a legal means and precedence for voluntary seeking of guardianship appointment. Thus, guardians could be appointed if a person was seriously ill or contemplating serious surgery, long-term hospitalization, or long-term absence from his family. The latter can be important in extended trips or overseas assignments of servicemen.

CONCLUSION

North Dakota law intimately ties the act of appointing a guardian and the declaration of incapacity. The constitution gives exclusive original authority to the county court, and the statutes describe necessary qualification for the appointment of a guardian as: a minor, habitual drunkard, spendthrift, or incompetent. The statutes further imply incompetency as the incapability of taking care of oneself or one's property.

Incompetency is a difficult and serious matter. We physicians must accept our legal responsibility to help the courts. Because of the nature of mental illnesses and because of the conflict between feelings and actions, we must be constantly alert for the person who may be unconsciously seeking help from us to have someone care for his person or his estate. His inappropriate actions may be the clue as to whether or not he is, in fact, unconsciously asking for help. This is especially true for those individuals who suddenly change their pattern of living, begin spending large amounts of money, or perform inappropriate actions. They are often caught in the horns of some dilemma and do not know how to verbally seek help or for some reason are ashamed to ask for help. These people need all the help we can give them at an early date before they have ruined their lives and developed a pattern of living unsatisfactory even to them.

We must not be too glib about our role and unconditionally state that a person's inappropriate actions are his way of seeking help for himself. We must consider that there are many ways of helping emotionally wrought individuals. The appointment of a guardian is not always the best. Civil and personal rights are too important to be dealt with haphazardly. Thus, even though the laws are ambiguous and laden with red tape, they serve as a means of protecting those who need protection. As Dr. Thomas Szasz states, "We have no right to this easy absolution from responsibility."[3]

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Circadian Rhythms and the Pineal Gland

R. E. WIEDERANDERS, M.D., K. A. LIND,
and G. W. EVANS, B.S.

Williston, North Dakota

If two systems show amplitude variations in time in an identical fashion, the temptation is great to look for a causal relation between them. Two such systems are the temperature cycle and the pineal gland activity in the rat. The temperature cycle in the rat and in virtually all other mammals is well known to vary with the circadian cycle.[1] Less well known is the recent work which shows that the rat pineal gland also varies with this cycle through its linkage to the lateral eyes by the sympathetic nervous system.[2]

In the rat, light stimulates 5-hydroxytryptophan decarboxylase activity to produce serotonin,[3] the midday level averaging about 90 nanograms per pineal. With the onset of darkness, serotonin is depleted at the rate of 25 nanograms per hour to a midnight low of 10 nanograms per gland.[4] This decrease is accounted for partly by conversion of serotonin to N-acetylserotonin and secondarily by O-methylation of N-acetylserotonin to melatonin via an enzyme peculiar to the pineal gland: hydroxyindole-O-methyl transferase (HIOMT).[5] Thus, melatonin is the peculiar product of this gland.[6] Serotonin production continues to build up and decline according to the circadian cycle in blinded rats and those kept in constant darkness but is abolished by constant light.[7] Darkness stimulates the activity of HIOMT and, thus, the production of melatonin, while light suppresses it.[8] This situation in the rat, a nocturnal animal, is reversed,[9] in the hen, a diurnal animal, serotonin being produced at night and melatonin during the day.

Suppression of the circadian cycle by continuous light is another point at which these two systems agree. As stated above,[7] the serotonin content of the rat pineal gland builds up to a daily high about 1:00 P.M. The 1:00 P.M. level stays high and the 11:00 P.M. level increases until it equals the earlier level if the animal is exposed to continuous light, thus obliterating the circadian rhythm. In temperature experiments on rats, Halberg[10] has found that darkness does not disrupt the circadian cycle, while continuous light throws it progressively out of phase and disrupts the cycle completely. The blood eosinophil rhythm which follows the circadian cycle also persists in the face of darkness but is disrupted by light.[11] Reserpine will alter the temperature cycle[12] and will deplete both the pineal gland and the brain of serotonin,[13] but the relation is confused, since acute reserpine administration depletes the brain but not the gland while chronic administration depletes both.[14]

It certainly seems that melatonin activity is highest when the animal is most active and the temperature highest. Therefore, the pineal may either regulate the temperature directly or form an intermediate link in its regulatory mechanism.

PROCEDURE

Twenty-nine locally raised Wistar rats were used, taken at random as to sex and held in 5 plastic cages. Twelve were normal, 13 were pinealectomized (PND) by our usual method,[15] and 4 were enucleated of their lateral eyes. The surgeries were performed at age 30 days and the temperature experiments begun at age 8 months. The laboratory had both natural and artificial light, so the light-dark cycles were variable. The temperature was controlled by air-conditioning but varied as much as 5° F. in any one day. All handling of the animals was done by one of us to introduce as little variation as possible. A Model 425C telethermometer* was used throughout. The 402* thermistor probe was used to take the four-hour and the fifteen-minute temperatures but, the 401* thermistor probe was used for continuous temperature monitoring. The telethermometer was connected to a Model 80 laboratory recorder* for this experiment.

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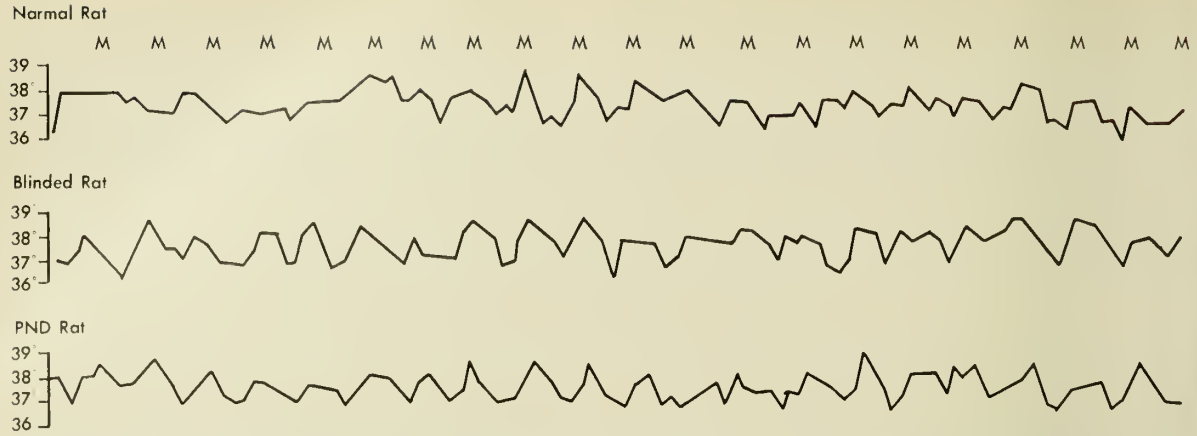


Fig. 1. Skeleton graph showing the rectal temperatures for representative rats of the 3 groups taken at four-hour intervals (except for 4:00 A.M.) for twenty-one days. Midnight is indicated by the dot under the "M"s.

Temperatures were taken on every rat at 5 four-hour intervals, beginning at 8:00 A.M. and continuing until 12:00 midnight. Each time the probe was introduced a distance of 4 cm. into the rectum and held for thirty seconds. Temperature was read to the nearest 0.2° C. and plotted. Readings were continued twenty-one days.

The second set of temperatures was taken on each rat at fifteen-minute intervals for two hours in the morning and two hours in the evening. Continuous temperature monitoring was done on 6 rats, 3 from each group for twenty-six hours beginning at 5:00 P.M. Small wire cages that would just hold the rat in a squeezed position were constructed for this, and the rats were held thus for the twenty-six hours. If the animal were allowed any leeway at all he would work around and bite off the probe. The data taken at four-hour intervals were handled by extracting the arithmetical mean for each of the 5 periods. Standard deviations and tests of significance were not used, as these data were negative. The fifteen-minute temperatures showed no regular pattern, and their only function was to lead us to the continuous temperature work. At the end of the experiment the animals were sacrificed to verify complete removal of the gland.

RESULTS

The raw data obtained from taking temperatures every four hours are shown in Figure 1 for 1 rat of each group. The "M"-points show the time of midnight and each sharp bend in the line represents a temperature period. Notice that the high points tend to fall at midnight, but all of them certainly do not, and there is great variation with each rat. However, each of these 3 animals tends to follow the same pattern. Collecting the 21 temperature values at each time period and establishing their means and plotting these together with the extremes resulted in Figure 2, in which representative animals from each group are again shown. Every animal studied showed the lowest temperature at noon

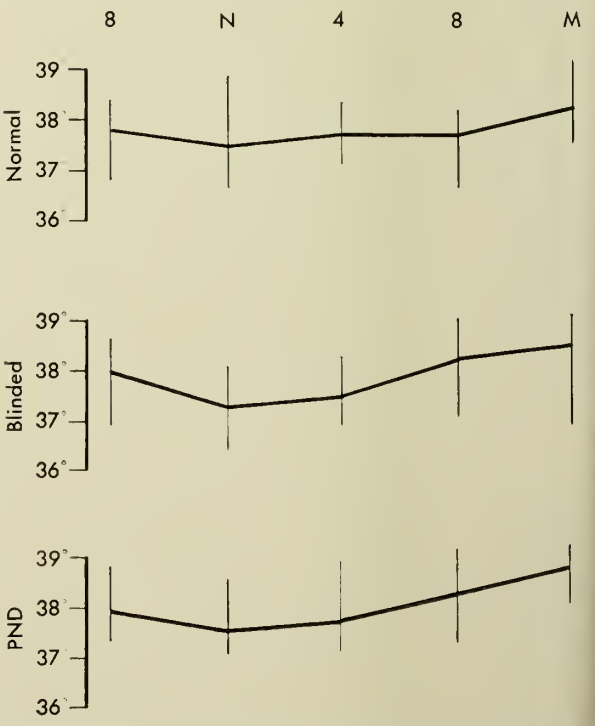


Fig. 2. Plots of the extremes, connected by the vertical lines, and the arithmetical means, connected by the horizontal line, of the temperatures taken every four hours on these rats for twenty-one days. Noon is indicated by "N" and midnight by "M." Each animal here shows a low at noon and a midnight high. All the rats in this experiment had exactly the same curves except for one blinded animal who had a high noon and a low midnight temperature. Notice the wide variance in extremes.

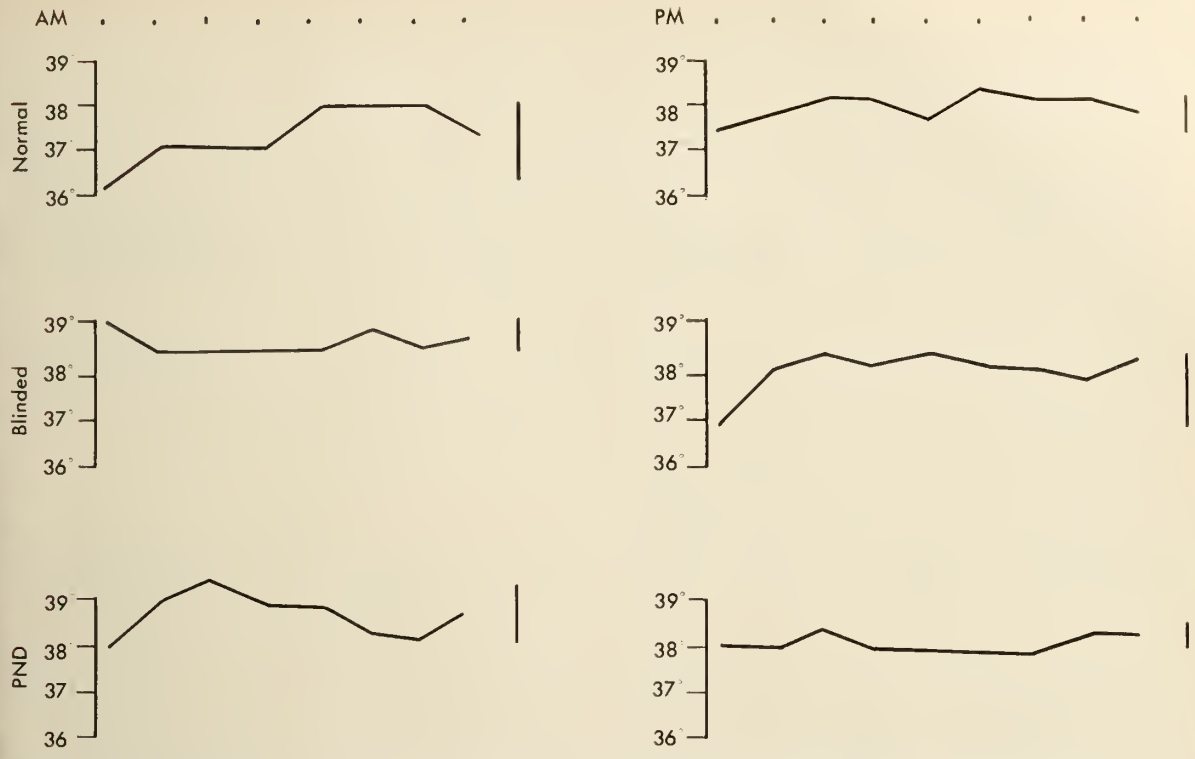


Fig. 3. Temperatures taken every fifteen minutes for two hours in the morning and again in the evening are plotted here. They show the temperature cycle of the rat to be complicated and led to the tracings shown in Figure 4.

and the highest at midnight, except for 1 blinded animal who showed the opposite. There was no difference in the normal and PND rats.

The extremes in Figure 2 are seen to vary widely, and this raised the question of just what type of temperature curve the animals had. To study it more closely, we took temperatures every fifteen minutes for two hours in the morning and again in the evening (Fig. 3). These showed very definite changes during these periods, indicating a more complicated curve and the need for continuous temperature monitoring (Fig. 4). The beginning of each graph indicates 5:00 P.M. and the recording was continued for twenty-six hours. The small numbers on every fourth vertical line mark off hours, but the numbers themselves do not refer to the particular hour that the temperature was taken. (If you wish to know this, take the number at the left as 5:00 P.M. and count by hours to the right.)

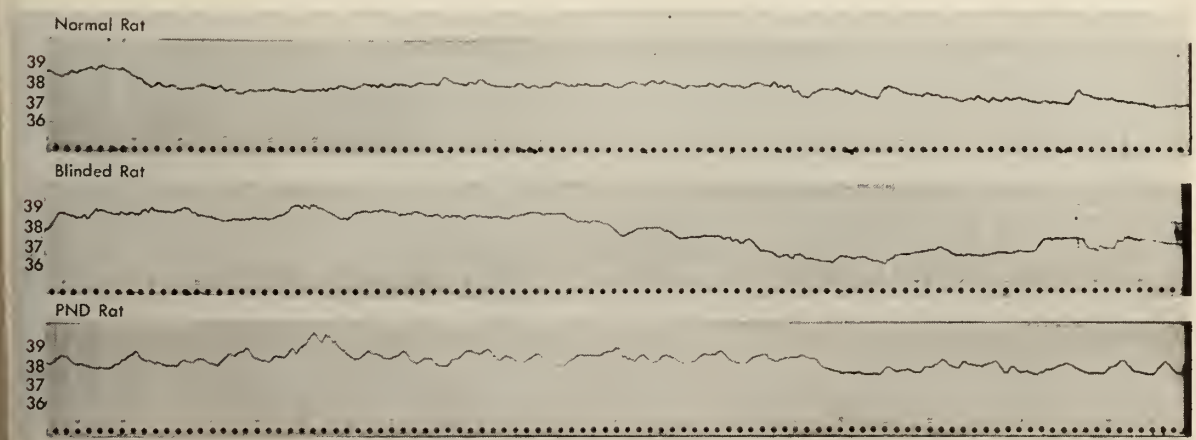


Fig. 4. Continuous temperature monitoring on 1 rat of each group produced these extremely complex temperature curves. Each curve represents a twenty-six-hour period.

When these graphs were completed, it was apparent that the temperature curve of the rat is extremely complicated and built of many interwoven harmonies. Each curve tends to be higher in the first half than in the second. The PND rat shows smaller variances of about one-half hour each, while the other 2 tend to vary over fifteen-minute periods with some longer periods as well. We have no idea what processes these fluctuations mirror or what significance they have. Looking at these tracings, one can readily see why the extremes in Figure 2 are so great. Taking temperatures at four-hour intervals leaves one totally dependent on chance as to what phase of which cycle one measures. The enucleated animals served as a second type of control. Since the lateral eyes affect the function of the pineal by way of the sympathetic nerves, this is a way of partly decycling the gland without removing it.

DISCUSSION

The reversal of cycle in the 1 blinded animal may be an example of cycle rephasing found in the mouse. The blinded mouse shortens his temperature cycle a small amount each day (forty minutes), reversing the cycle at twenty-two days and regaining phase at thirty-six days.[16,17] This is the only alteration in cycle shown by these experiments. The normal and PND animals have precisely the same temperature curve and so do 3 of the 4 blinded animals. Since all these animals react in precisely the same manner, it seems safe to say that the pineal gland does not control the body temperature or form a link in the pathway of such control.

Rather, it is, itself, controlled by the same oscillator that controls all the circadian cycles, located, perhaps, in the dienecphalic-hypophyseal system.[18] This is responsible for the daily build-up of serotonin and the nightly fall. The elaboration of melatonin is controlled differently, requiring light as its "Zeitgeber" to entrain it to the environment.[19] Possibly light adjusts the serotonin cycle to hold it to that of the environment as it does with the temperature cycle of the mouse. The pineal serotonin, then, is controlled by another structure. But what of the serotonin in the brain itself? As mentioned above, its level is decreased by reserpine, which also abolishes the circadian rhythm of the temperature cycle. Does the brain serotonin content vary in a circadian fashion in response to this mysterious oscillator? Does this serotonin play a role in temperature regulation which is abolished when its level is low?

SUMMARY

Because the pineal gland and the body temperature function according to a circadian cycle, a correlation was sought between them by taking periodic temperatures on normal, pinealectomized, and blinded rats. No alteration of the temperature cycle was present in any of these animals except for one blinded rat, showing that the pineal gland is not concerned with temperature regulation.

Wide extremes of daily temperature prompted further investigation. Continuous temperature monitoring showed the rat to have several cycles of greater and lesser length which combine to form a very complicated picture.

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Acute Bronchiolitis as a Cause of Chronic Obstructive Lung Disease in Adults

Report of Two Cases

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TWO ADULT PATIENTS have recently been seen at the Mayo Clinic with chronic obstructive airway disease which developed after acute upper respiratory tract infection suggestive of obstructing bronchiolitis. There has been little published concerning the clinical significance of primary bronchiolitis in the adult or about the degree of reversibility afterward.

REPORT OF CASES

Case 1. A 48-year-old nurse was seen for the first time in December 1965 because of progressive exertional dyspnea. She had never smoked. She had been in good health until one year before when she had an acute viral infection of the upper part of the respiratory tract with cough, fever, and purulent sputum. She had moist rales and wheezes and was hospitalized for seven days. She had no previous history of allergy and no family history of allergy. She had progressive shortness of breath, wheezing, and cough and had responded poorly to treatment over a ten-month period.

On examination here, she was dyspneic at rest. She had marked expiratory slowing. The thoracic roentgenogram, electrocardiogram, and results of routine laboratory tests were normal. A sputum culture grew out a normal flora. Bronchoscopy, performed because of the abrupt onset and the lack of antecedent allergic history, showed subacute and chronic bronchitis. Ear, nose, and throat examination gave normal findings; roentgenograms of the sinuses were normal.

Pulmonary function studies (Table) showed a pattern of chronic obstructive lung disease with reversibility after treatment with nebulized isoproterenol.

Case 2. A 53-year-old man, an executive, was seen in December 1965, three days after the first patient. He had been in excellent health until October 1965 when he had had an acute viral upper respiratory tract infection with fever, cough productive of sputum, and wheezing. He had fine and medium rales throughout both lungs. He had never wheezed before the acute infection. He was hospitalized and treated with intermittent positive-pressure breathing and antibiotics. He had no limitation in his exercise tolerance until after this infection. Since, he had experienced progressive exertional dyspnea, cough, and wheezing and had been unable to put in a full day's work. He had no history of allergy and no family history of allergy.

The findings on physical examination were normal except for the expiratory slowing and inspiratory and expiratory wheezes. Ear, nose, and throat findings, thoracic and sinus roentgenograms, electrocardiogram, and results of routine laboratory tests were normal. A sputum culture grew out a normal flora. Pulmonary function studies (Table) showed a pattern of obstruction with reversibility after treatment with nebulized isoproterenol.

DISCUSSION

The pathogenesis of emphysema remains obscure.[1] Although the mechanism is not clearly understood, there is little doubt that infection is a factor in the pathogenesis, but whether viral bronchiolitis is also an etiologic factor is not yet known. Respiratory syncytial virus has caused bronchiolitis in children, with the clinical picture of generalized obstructive airway disease.[2] McLean[3] thought that the most significant etiologic factors in emphysema were related to inflammatory damage of bronchioles, but Smith[4] found no significant association between acute bronchiolitis and emphysema of any type in a purely anatomic study. Ham[5] found acute, infectious, obstructing bronchiolitis in 7 adults; the obstruction was due to various combinations of bronchiolar infection, bronchiolar spasm, inflammation and thickening of the bronchiolar walls, and plugging of the bronchiolar lumen by purulent mucous plugs.

Bronchiolitis can be diagnosed clinically by the presence of cough, fever, wheezing, fine and medium rales, and a prolonged expiratory phase. In both of our patients the clinical picture of

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acute, diffuse, ventilatory obstruction developed secondarily to an apparently benign, acute, nonspecific, respiratory tract infection. It would appear that the primary pathologic process was acute, infectious bronchiolitis, most likely viral. McLean[3] postulated that inflammatory bronchiolar disease can occur from acute viral infections, acute bacterial infections, or bacterial infections secondary to viral infections. He indicated that primary bacterial bronchiolitis is rare, whereas bacterial infection secondary to viral bronchiolitis is extremely common.

Bronchiolitis occurs mainly in young children, but it does occur in adults[6] and may develop into chronic, obstructive lung disease.[5] Recurrent infections with progressive obliteration of more and more bronchioles can result in generalized irreversible bronchiolar obstruction. Persistence of bronchiolar obstruction prolongs the phase of air trapping even after the exudative phase of bronchiolitis. It seems reasonable that an acute viral infection of the respiratory tract in an adult could set up diffuse bronchiolar obstruction with spasm. Haemophilus influenzae may be a secondary invader at this phase. Combinations of chronic spasm, superimposed bacterial infection, inflammatory and thickened walls, and purulent exudate in the lumina produce permanent, irreversible, obstructive airway disease.

Treatment[7] must be initiated early in the course of the disease to prevent plugging of the bronchiolar lumina. A high fluid intake, administration of potassium iodide and bronchodilators, intermittent positive-pressure breathing with isoproterenol by nebulization, and use of broad-spectrum antibiotics are indicated. Oxygen therapy during the acute stage may be lifesaving if the patient is hypoxic. Bronchiolitis can be mistaken for acute bronchitis, congestive heart failure, or bronchopneumonia. Early recognition and vigorous treatment are essential.

SUMMARY

Two adult patients with chronic obstructive airway disease occurring after acute upper respiratory infection suggestive of obstructing bronchiolitis have been described. Although more common in children, acute bronchiolitis can occur in adults. More work is necessary to determine whether a virus by itself can produce obstructive bronchiolitis or whether the viral infection has a superimposed bacterial component. Treatment must be initiated early and be vigorous to prevent an irreversible, chronic, obstructive process.

(References on page 295)

RESULTS OF PULMONARY FUNCTION STUDIES

	Case 1			Case 2		
	Estimated normal	*Without isoproterenol*	*With isoproterenol*	*Estimated normal*	*Without isoproterenol*	*With isoproterenol*
Pulmonary volumes						
Vital cap. (exp.) (L.)	3.3	2.03	2.52	5.6	4.10	4.05
Insp. vital cap. (L.)	3.3	2.12	2.68	5.6	4.22	——
Residual vol. (L.)	1.3	2.47	——	1.9	3.24	——
Total cap. (L.)	4.6	4.58	——	7.5	7.46	——
RV per TC × 100 (%)	<35	54	——	<40	43	——
Functional residual cap. (L.)	2.1	3.10	——	3.4	3.87	——
Exp. reserve (L.)	0.8	0.63	——	1.5	0.64	——
Max. breathing cap. (L. per min.)	81	51	89	122	89	116
Max. midexp. flow (L. per sec.)	>2.0	0.7	0.9	>2.0	1.2	1.5
Exp. slowing (grade)	0	3	3	0	2	1
N_2 washout index (%)	≤2.5	4.2	——	≤2.5	0.9	——
Arterial O_2 sat. (%)						
Resting (air)	95-98	94	——	95-98	99	——
Resting (O_2)	100	100	——	100	100	——
Before exercise (air)	95-98	95	——	95-98	99	——
Exercise (air), 5 min.	95-98	96	——	95-98	99	——
Ventilation (sitting at rest, O_2)						
L. per min.	——	11.10	——	——	13.00	——
Resp. per min.	——	17.8	——	——	12.0	——
Average tidal vol. (ml.)	——	624	——	——	1,083	——

Section on PAIN *and* DISTRESS

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The Mystery of Atypical Pain

The section on Pain and Distress in *The Journal-Lancet* is published in the hope that it may bring enlightenment to the mystery of atypical pain and distress. The current article, "Cerebral Vasospasm in Migraine," by Dr. Donald J. Dalessio, fits into this effort and is reproduced for its thought-provoking possibilities. Discreet and recognizable lesions of the central nervous system produce a variety of symptoms, and some of these may be imitated by unfamiliar phenomena. In the past, attempts to identify circulatory disorders as the cause of pain and distress have been difficult.

Encouraging progress may now be available using new medications as a help in differential diagnosis. It is hoped that this is an interaction of time and effort by the pharmaceutical industry and the physician that has been long awaited.

J. S. LUNDY, M.D.

Cerebral Vasospasm in Migraine

DONALD J. DALESSIO, M.D.

La Jolla, California

THERE IS ABUNDANT evidence for the existence of a nerve supply to the dural, pial, and intracerebral blood vessels of man.[1] Recent evidence suggests that this nerve supply may be more complex than previously determined. Using the electron microscope, Dahl and Nelson[2] have demonstrated myelinated and unmyelinated nerves in the adventitia of the superior cerebellar and posterior inferior cerebellar arteries of twenty brains sectioned. Neurophysiologists have not, however, been convinced that neural vasomotor effects are of importance in the regulation of the cerebral circulation. Thus, according to Sokoloff and Kety,[3] ". . . while it is clear that neural vasomotor mechanisms exist, at least under laboratory conditions, . . . their role in the regulation of cerebrovascular tone is obscure." In short, it is postulated that, if the blood pressure remains constant, the tone of the cerebral blood vessels is most profoundly influenced by the relative tensions in the blood of the respiratory gases, oxygen and carbon dioxide.[4]

Clinical observations indicate, however, that the large arteries of the brain may be capable of active and pronounced vasospasm independent of changes in the respiratory gases. Vasospastic arterial responses have been documented by direct observation during neurosurgical procedures and by radiographic studies employing angiographic techniques.[5,6]

The clinical and experimental observations on the occurrence of cerebral vasoconstriction have been summarized in a series of articles by Pool.[7] It is his conclusion that cerebral vasoconstriction is mediated by the musculature of the vessels in response to variegated local stimuli. He also infers that a central control of vascular tone may exist.

In general, the clinical or angiographic observations on cerebral vasospasm previously men-

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tioned have been related to intracerebral catastrophes, such as subarachnoid hemorrhage, or to manipulation of the cranial blood vessels during neurosurgical procedures. This is not to say that these episodic excesses of cranial vascular spasm do not occur in more benign circumstances.

There is, for example, good experimental and clinical evidence that migraine is characterized by profound alterations of both intracranial and extracranial blood vessels, of both constrictor and dilator nature. It has been stated by Wolff[8] that the initial, painless, preheadache experiences of migraine, including especially scotomata and field defects, are directly related to the vasoconstriction of the cranial blood vessels. In some fashion, this initial brief period of vasoconstriction is replaced by a prolonged phase of distention and dilatation of the cranial arteries; that is the painful stage of migraine which may then be relieved by vasoconstrictor agents including the ergot alkaloids and norepinephrine.[9] The vasospastic episodes which characterize migraine may not always be innocuous, however. Connor[10] has described eighteen patients with lesions of the retina, cerebral hemispheres, and brainstem which he postulates were directly related to repeated episodes of migraine. Others have made similar observations, usually involving the occipital cortex and often producing field defects of cortical origin.[11]

Wolff and Guest[12] describe in detail an episode of fatal infarction of the brain in a 28-year-old male with recurrent migraine attacks. No other structural abnormality of the cerebral vasculature or heart was demonstrable. The cause of death was ischemia of the brainstem. The authors suggest that the disturbance of cerebral blood flow involved more than one of the intracerebral arteries.

Williams and Wilson,[13] in a study of basilar artery insufficiency, state that migraine attacks appearing in late middle life may constitute a harbinger of subsequent and severe cerebrovascular insufficiency, especially of those syndromes associated with decreased blood flow through the vertebrobasilar system. Bickerstaff[14] has described episodes of migraine in which the basilar artery was particularly involved.

Heron reports seven patients in whom migrainous symptoms and neurological complications are described. Three of these patients died. Their mean age at onset of symptoms was 40 years, and the average duration of symptoms was ten years. Heron notes that the signs and symptoms of hemiplegic migraine and cerebrovascular insufficiency may be identical. He suggests that vasoconstriction in the larger intracranial or extracranial vessels may be an explanation for symptoms and neurological defects occurring not only in hemiplegic migraine but in some cases of proved primary cerebrovascular disease with atheromatous lesions.[15]

It is evident that the basilar system, supplying as it does the brainstem and the central connec-

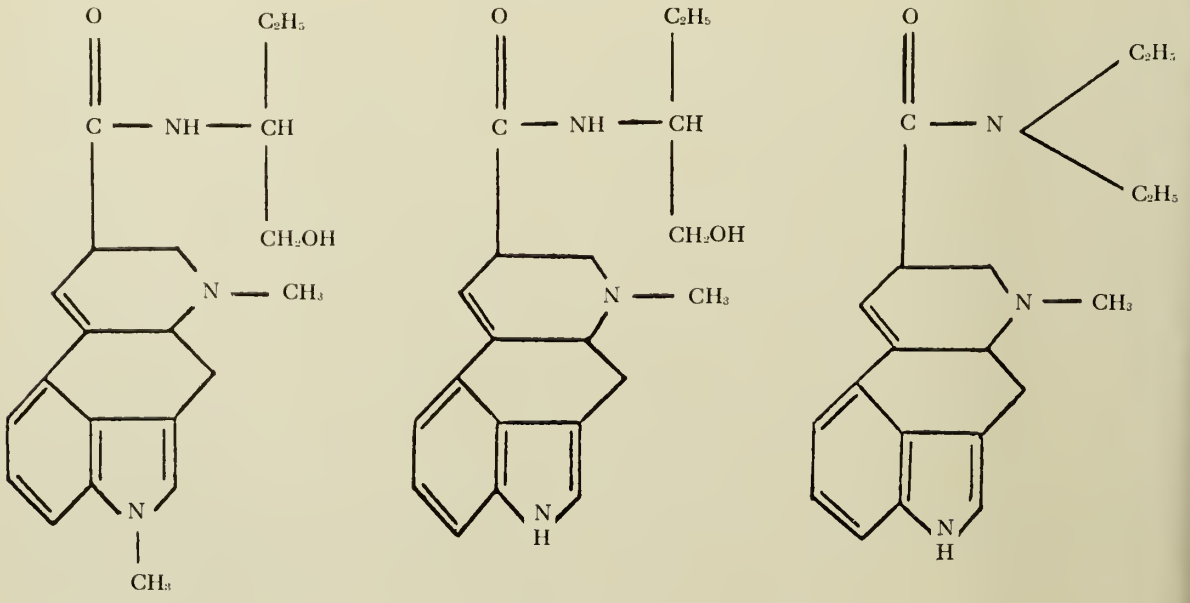


Fig. 1

tions of the organs of sight and hearing, occupies an earlier site on the phylogenetic scale than do the carotid vessels, whose main purpose is to nourish the neopallium. Furthermore, many of the premonitory signs of migraine, including field defects of cortical origin, nausea, vomiting, vertigo and unsteadiness, and retention of salt and water, can be construed as relating to basilar arterial spasm.

Some studies with the lysergic acid derivative, methysergide, are of interest with respect to the reactivity and tone of the cerebral blood vessels. Methysergide (1-methyl-D-lysergic acid butanolamide) differs from the hallucinogenic agent lysergic acid diethylamide only in that it contains a butanolamide rather than a diethylamide side chain. It is intimately related to the naturally occurring ergot alkaloids, especially ergonovine and its derivatives (Fig. 1).

Methysergide has acquired a certain clinical usefulness in the treatment of vascular headache of the migraine type. It has been termed effective by several investigators in the prophylaxis of headache, although it lacks the capacity to terminate a headache already in existence.[16]

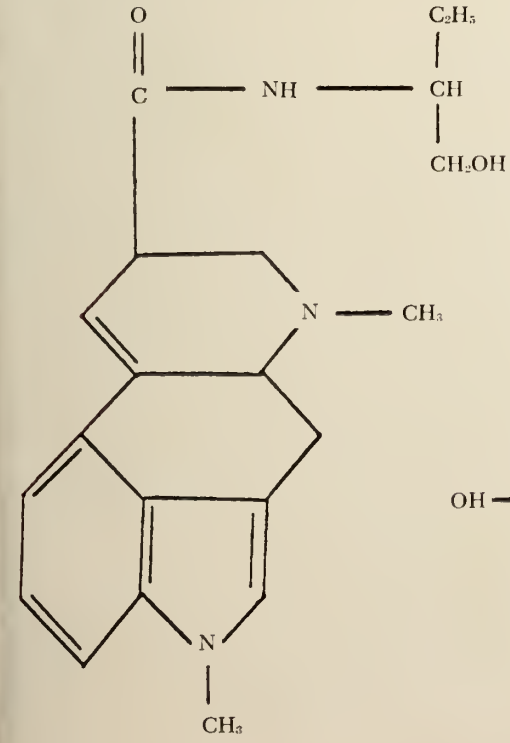
The compound will potentiate the vasoconstrictor properties of the catecholamines, other ergot derivatives, and nicotine.[17]

Sicuteri has shown that methysergide will exert a peripheral and protective effect against the hyperalgesia produced by serotonin.[18] He has demonstrated further that methysergide will protect against cutaneous, vasomotor, and cephalgic reactions caused by the histamine liberator 48/80. These peripheral reactions of methysergide may be of great importance in the prophylaxis of migraine. In addition, it has been shown to be effective in modulating the central vasomotor functions of both men and animals, with a consequent decrease in cranial vascular reactivity and a reduction in the frequency and severity of headaches.[19,20]

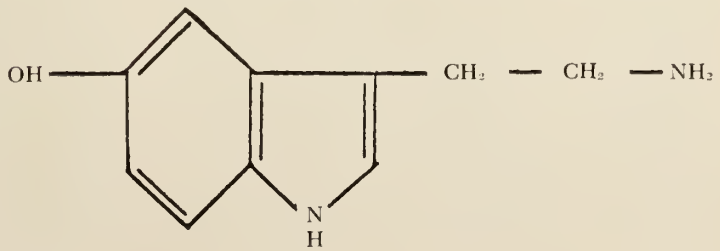
The mechanism by which methysergide exerts its effects on the central vasomotor centers is unknown. Among its more interesting properties, however, is the capacity of methysergide to inhibit serotonin (5-hydroxy-tryptamine).[21] This inhibition is competitive and presumably related to a similarity of structure (Fig. 2).

These data are of interest with respect to the function of the central vasomotor centers. In a sense, these centers function as "head ganglia" of the autonomic nervous system. Brodie and associates,[22] utilizing the concepts of Hess,[23] have suggested that serotonin may function as the neurohormone of a parasympathetic subcortical system which integrates the autonomic nervous system with the remainder of the brain. (These authors further suggest that norepinephrine may be the hormone of a competitive adrenergic system of a similar sort.) Furthermore, it has been demonstrated that concentration of brain serotonin is intimately related to the functions of the central vasomotor centers in animals.[24,25] It is at least worthy of speculation, then, to suggest that the antiserotonin action of methysergide may be associated with its ability to modify the functions of the central vasomotor centers.[19]

Migraine may thus be viewed as an episodic, usually, but not always benign, vascular spastic process, involving both the intracranial and ex-



Methysergide



Serotonin

Fig. 2

traeranial blood vessels and including eonstrictor and dilator phases. A serotonin antagonist, methysergide, has proved to be effective in the prophylaxis of this disease. Furthermore, when effeetive, methysergide prevents both the preheadaehe or vasoeonstrietor phase and the headaehe or vasodilator phase of migraine, possibly by altering the aetivity of the eentral vasomotor eenters, and so diminishing the exeessive aetivity of the eranial blood vessels, and by inhibiting the elaboration and aetion of vasoneuraetive substanees as they are eharaeterized by Sieuteri.[18]

These data suggest that the eentral vasomotor eenters do perform a funetion in the regulation of the eerebral vaseular tone. They suggest that the eentral autonomie nervous system, though a nerve supply already demonstrated in anatomical studies, does have a role in the regulation of segmental eortieal blood flow. (It may perhaps beeome evident when more precise methods for evaluating loeal cerebral blood flow in man become available.) And finally, they provide a elue to the pathophysiology of one sort of eerebral vasospastie episode.

These eoneepts are not in opposition to the monumental work on total eerebral blood flow whieh has already been aeeomplished. It is obvious that the eerebral blood vessels are exquisitely sensitive to the level of respiratory gases in the blood and that the major determinant of cerebral vascular tone is chemical and not neurogenie.[4] It is suggested, however, that *not all forms of human cerebrovascular disease* can be comfortably assimilated under the previous general physiological law. Further studies on the neurogenic control of the great vessels at the base of the brain are neeessary, espeeially of the phylogenetieally older vertebrobasilar system.

CONCLUSION

The large arteries of the base of the brain may be eapable of aetive vasospasm. This tvpe of exeessive eranial vaseular reaetivity is often asseeiated with intraeerebral eatastrophes but may oeeur in more benign states, sueh as migraine.

It has been demonstrated that methysergide, a lysergie acid derivative, will prevent migraine and that the prophylaetie qualities of this medieation are related, at least in part, to its ability to alter central vasomotor funetions. The relationship of brain serotonin to the funetion of the central vasomotor eenters is diseussed.

These studies suggest that the eentral vasomotor eenters and the eentral autonomie nervous system may perform a neural regulatory role in the control of the eerebrovaseular tone of the major blood vessels at the base of the brain and, especially, of the vertebrobasilar system.

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Control of Pain in Patients with Cancer

GUY OWENS, M.D.

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On occasion, laymen, as well as physicians, inquire of neurosurgeons how they can practice medicine with equanimity when their patients have so high an incidence of incurable diseases and sudden and untimely deaths and there are, therefore, so few therapeutic measures available. These obvious points are best neutralized by stating that one of the most satisfying experiences for a neurosurgeon is the surgical relief of intractable pain. Often pain is associated with a benign cause. A high percentage of patients, however, are afflicted with an incurable malignant disease. Once again, it is not uncommon for others to comment on how discouraging this must be. Not at all. Pain, whoever experiences it, becomes the focus of one's life, however long that may be.

Because of the increasing frequency of malignant disease and because of a longer life expectancy brought about by improved therapeutic management, many people are now surviving to experience severe incapacitating pain during the terminal period of their lives. Though pharmacologic control of pain is adequate to handle most problems, we have been dissatisfied with medication as a permanent solution. An important factor in considering the management of intractable pain, especially in those afflicted with malignant disease, is the economics of further surgery. At Roswell Park Memorial Institute a patient can accept or decline such efforts for reasons other than economic. The attitude of the following remarks, therefore, is heavily weighted by surgical concepts. It is our belief that pain caused by a malignant disease deserves as much attention as pain from a ruptured appendix in a patient with an incurable malignant disease.

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NONSURGICAL CONTROL OF PAIN

In our institution other clinical departments serve as triage centers, excluding many problems which can be handled conservatively (nonsurgical). This triage service has one disadvantage. Occasionally, patients could have profited by earlier surgical intervention.

Pharmacologic control of temporary pain is a boon to medicine. It becomes a hazard when pain becomes intractable. In the initial stages of a malignant disease, direct measures to irradicate the tumor should be taken. This may relieve the associated pain. It is after this therapy has failed that more and stronger medication is required. Analgesic control of pain becomes impossible, and graduation from codeine to meperidine (Demerol®) to morphine occurs. Most people under such conditions assert that they are never free of pain even when narcotized to the point of sleep. It is at this time that neurosurgical advice is usually sought. Even for the neurosurgeon, it becomes difficult to persuade the patient and his relatives that major surgical intervention is indicated. It is tempting to try nerve blocks, radiation therapy, or chemotherapy instead of surgical operation.

Peripheral or subarachnoid nerve blocks, using absolute alcohol or phenol, have an important place in handling intractable pain. These techniques are limited by the accuracy of percutaneous location of the appropriate sensory unit and by the risk of unwanted diffusion of the neurotoxic solution. A 5% solution of phenol in glycerin has proved to be an acceptable agent; 0.75 cc. of this solution may be placed in the subarachnoid space so that the toxic phenol comes in contact with the posterior nerve roots. It has been shown that there is a general, though limited, destruction of all fibers whether motor or sensory. The work of Maher[1] in England and of

several groups in the United States has indicated that it is associated with frequent side effects and is successful in approximately one-third of the situations employed. Others have noted that the results are transitory.

We have been unimpressed by radiation therapy as a permanent solution to pain problems when the tumor is known to be insensitive to X-ray (bronchogenic, bowel, bone prostate, cervical uterine, and, occasionally, breast). On occasion, spinal cord compression has been neglected while therapy has been directed toward pain control.

More recently, chemotherapy has been explored as a tool for specific pain therapy as well as oncolysis. Its value in Hodgkin's disease and other lymphomas is well recognized. Some value in isolated arterial perfusion of an alkylating agent has been observed. It has been shown that pain relief thus afforded may come about by direct damage to the peripheral nerve or sensory nerve ending.[2] The perfusion technique is not considered conservative nor is it always associated with permanent relief of pain. Prolonged intra-arterial infusion of chemotherapeutic agents may offer further advantages in the future.

Unfortunately, the above efforts frequently come to naught. Time, meanwhile, has passed and the patient has become addicted. Often following surgical relief of pain the patient is outspoken and critical of himself for agonizing over the necessity for further surgery. We have found that group therapy is helpful in allowing a patient to arrive at a decision to accept pain surgery. Patients who have had similar surgery can act as counselors as well as by example. In no other surgical endeavor does the threat of the unknown seem to pose such a problem to the patient.

During the past seven years at Roswell Park Memorial Institute, education by example has increased the referral to the neurosurgical service. This process of education has been important to the neurosurgeon also. It has required frank recognition and discussion of the undesirable effects of the surgical effort. It has led to a clearer recognition of the type of procedure required in certain situations. It has produced investigative efforts which have corrected undesirable complications.

SURGICAL RELIEF OF PAIN

Since pain is entirely subjective we have found that the physician cannot afford to sell his surgical efforts too vigorously. A dissatisfied and iatrogenically disabled patient can be most unpleasant. It has been our policy, therefore, to describe the various procedures in detail to the patient including the undesirable, but often observed, side effects. Under these circumstances, he must understand fully the degree and seriousness of his underlying illness. He can then ask for help with full understanding of the undesired costs. We find a usually highly satisfied person after surgery. This also is the best measure of the severity of his pain. Those people who fear the aftereffects of surgery are still able to obtain pain control through other means. Some even feel that neurosurgical control of pain has now reached the point where its risks and possible complications are less than those of narcotic addiction. Operative intervention for relief of pain in malignant disease is most desirable in patients with a life expectancy of six to twelve months. It is very difficult, however, to predict the future and for that reason we have offered procedures to some patients who have not survived as long as hoped.

During the past several years a new technique, the high cervical percutaneous cordotomy, has permitted stereotactic nonsurgical destruction of anterolateral spinothalamic tracts. Employing this method, life expectancy is not so important because the aftereffects are not much more serious than those experienced following a lumbar puncture.

Neurosurgical procedures include cordotomy (operative and percutaneous), rhizotomy, decompressive laminectomy, lobotomy, and hypophysectomy.

Rhizotomy. Section of the posterior or sensory cranial and spinal nerves can produce anesthesia in a well-defined region. Because all sensory modalities are thereby affected, this procedure should not be employed in a useful extremity. Total anesthesia in the thorax and abdomen is inconsequential and, therefore, very valuable. The major difficulty is that an extensive laminectomy may be necessary. This operation, however, is usually well tolerated. A temporary block with procaine can often delineate very accurately the nerve root or roots to be cut. Sections of the fifth, ninth, and tenth cranial nerves can often give good control of pain in the head and neck cancer problems. A combination of cordotomy and rhizotomy can often be effective in shoulder and upper extremity pain.

Prefrontal lobotomy. This procedure has been reserved for the desperately ill when pain can no longer be controlled. The operation consists of bilaterally sectioning the white matter in the medial quadrant of the frontal lobes. It is done under direct vision employing local anesthesia. The patient is immediately ambulatory if his

preexisting disease permits. In general, however, we are not satisfied with the sensorial side effects and do not promote this procedure actively.

Decompressive laminectomy. A careful neurologic evaluation of all problems must be conducted. This is especially true with pain in the lower extremities. This pain may be the initial manifestation of an intracannular tumor which produces pain by expansion and compression of the cord or cauda equina. Relief of pain under these circumstances may be effected by a decompressive laminectomy. Depending on the pathology, progression of the tumor may occur despite all therapeutic efforts.

Hypophysectomy. This is a special procedure in that it can be successfully employed only in patients with tumors which are expected to be hormone-dependent. Thus far, only patients with breast or prostatic carcinoma have been offered this procedure. It should be noted that bilateral adrenalectomy also is available and is actually the most frequent effort employed in this institution for therapy of advanced breast disease. The general experience has been that either approach, hypophysectomy or adrenalectomy, is equally successful. About 40% of all patients will respond favorably to hormone surgery.[3] After hypophysectomy, thyroid extract, cortisone, and, on occasion, vasopressin (Pitressin®) are necessary. Only cortisone is required after adrenalectomy. Craniotomy may be the best surgical route when abdominal surgery is deemed too debilitating. Complications from hypophysectomy include unilateral visual loss and, occasionally, seizures. Either of these two problems are generally avoided, however, by the use of urea and drainage of cerebral spinal fluid to reduce the intracranial brain volume. Mortality figures in large series of hypophysectomies range around 4%.[4]

Employing the transnasal transphenoidal anterior approach to the pituitary gland, it can be destroyed using a cryosurgical cannula placed in position stereotactically. This procedure is performed under local anesthesia and is well tolerated by the patient but can be associated with meningitis or cerebrospinal fluid rhinorrhea. Total destruction is now measured by assay of growth hormone.

The pain in breast or prostate disease which responds to hypophysectomy is due to involvement of bone. The mechanism for the relief is not understood but probably is related to a change in pressure produced by a resolution of the tumor within the bone structure.

Cordotomy. Sectioning of the anterolateral spinothalamic tracts which lie in the anterior quadrants of the spinal cord is the most effective and useful procedure now available for surgical control of pain. Temperature, as well as pain, is transmitted by way of this relatively well-defined pathway.

For the past seven years, cordotomy has been employed most frequently as a surgical means of relieving pain. Our patient population has ranged from 22 to 77 years of age. In 102 patients subjected to a unilateral or bilateral procedure during this period, pain had been present on an average of eight months. Selection of a patient for a surgical cordotomy must include an attempt to interpret the advanced nature of the underlying disease and, therefore, his life expectancy. It is very difficult, however, to predict the future so that we have offered surgical procedures to some patients who have not survived as long as was hoped. A cordotomy is best used when pain is localized to a useful extremity in which a rhizotomy would render it useless. We feel it is the procedure of choice when midline abdominal or pelvic disease and pain either unilateral or bilateral is present. In these circumstances we have elected to do a bilateral simultaneous low cervical cordotomy. We have learned that unilateral procedures performed in the face of pelvic or abdominal disease invariably are followed by pain on the opposite side. Bilateral simultaneous interruption of the anterolateral spinothalamic tracts will cause loss of bladder function in each instance. In our series, 65% of female and 22% of male patients recovered function sometime after surgery. We have no clear understanding of the intraspinal circuiting of the urinary bladder. Impotence can occur after cordotomies in males. Hypotension has been present to some degree in all patients subjected to bilateral procedure but no permanent undesired effects have been recorded. Weakness of the lower extremities has been observed in all patients. This has been transitory in all but three. If preoperative leg weakness existed on the basis of plexus or peripheral nerve involvement, increased weakness was always found postoperatively.

Selection of the level at which the anterior quadrant of the cord is sectioned is important. Bilateral division of the spinothalamic tract at C-1, C-2, or C-3 is associated with severe respiratory disturbances.[5] Anesthetic levels following cordotomies can be expected to drop several segments after edema subsides. For that reason, efforts are made to section the tract at as high a level as is practical.

More recently, using the percutaneous high cervical method developed by Mullan and as-



After a C-2 left anterolateral spinothalamic cordotomy, pain and temperature sensation were destroyed in the region outlined by the triangles.

sociates[6] and perfected by Rosomoff and coauthors,[7] we have learned that a new era of pain control in cancer patients has appeared. This technique permits the destruction of the spinothalamic tract simply by the stereotactic insertion of a spinal needle laterally between C-1 and C-2. A wire electrode is then inserted into the anterior quadrant and a lesion is made using a radiofrequency generator. Rather exact control of the lesion size makes it possible to follow stepwise the anesthetic level. The procedure can be carried out in terminal cancer states since minimal morbidity is experienced. Bilateral procedures are staged with a satisfactory sparing of bladder function. The procedure can be repeated if the anesthetic level falls or if pain recurs at a higher level. We have used this approach in twenty-five patients to date. Seven have been bilateral. No motor complications have been observed. Bladder dysfunction has persisted in two males. We have not observed respiratory disturbances but none have had anesthetic levels brought to the upper cervical region bilaterally. It is only at this point that we consider the entire quadrant destroyed. We have had one failure. One patient found the experience sufficiently unnerving that he requested the operative approach for the second side. It should be recognized that, occasionally, during the use of the radiofrequency current a burning sensation in the neck can become quite unpleasant.

The following is a brief summary of a pain problem in a patient with bronchogenic carcinoma.

CASE REPORT

K.E.M., a 46-year-old white male had a four month history of pain in the region of the right scapula with radiation into the right shoulder and upper arm. He had required codeine, Demerol, and barbiturates for a period of three months and no longer received even temporary relief. X-ray examination of the chest revealed a destructive lesion of the fourth rib on the right at the costovertebral junction. A needle biopsy of this lesion was obtained and a diagnosis of bronchogenic carcinoma was made. Because of the intractable nature and location of his pain, the neurosurgical service advised a left high unilateral cervical cordotomy. Pulmonary function studies were essentially normal. On Oct. 29, 1964, this procedure was carried out. The figure indicates the extent of his postoperative loss of pain and temperature sensation. He was discharged on Nov. 20, ambulatory, free of pain, and without unwanted significant postcordotomy neurologic deficits.

To date, a total of 127 patients have had pain relieved by division of the spinothalamic tract. Eighty percent have obtained objective pain relief. The subjective value vis-à-vis the surgical experience, temporary or permanent morbidity, and short duration of life afterward cannot be measured.

We feel that the majority of our patients subjected to surgery have benefited in some fashion. There are fewer pharmacologically induced side effects, such as lethargy, loss of appetite, and addiction. Movement becomes freer. A factor of some importance is improvement of the psychologic outlook once the pain is relieved.

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Dr. Ralph Moore Tovell

DR. RALPH M. TOVELL of Hartford, Conn., who gave me considerable assistance in the conduct of this section of THE JOURNAL-LANCET, died in the Hartford Hospital on Jan. 7, 1967, of a heart attaek.

Dr. Tovell was born Dee. 31, 1901, in Sydenham, Ontario, Canada. He attended the Sydenham High Sehool for five years and then enrolled in Queen's University in Kingston, from whieh he reeeived the degrees of Bachelor of Arts in 1924 and Doetor of Medicine and Master of Surgery in 1926. He was an intern in the Kingston General Hospital from June 1926 to June 1927, and from July to November of that year he was an assistant in general anesthesiology to Dr. E. J. MeKesson of Toledo, Ohio. From November 1927 to April 1928 he was a resident in the Lying-In Hospital of the City of New York, and from July 1928 to January 1929 he was in general practiee in Sydenham, Ontario.

Dr. Tovell came to Rochester, Minn. on Jan. 1, 1929, as an assistant in anesthesiology in the Mayo Graduate School of Medieine, in March he was appointed a first assistant in anesthesiology, and in October he became my colleague in the Section of Anesthesiology of the Mayo Clinie. He was appointed an instructor in anesthesiology in the Mayo Graduate School of Medicine in 1930 and was advanced to assistant professor in 1935.

Dr. Tovell resigned from the staff of the Mayo Clinie and Mayo Graduate School of Medicine on Sept. 1, 1936, to become chief of anesthesiology in the Hartford Hospital at Hartford, Conn., a post he retained until 1963. He also became consulting anesthesiologist to the following Conneeticut hospitals: Manchester Memorial Hospital, the Middlesex Memorial Hospital of Middletown, the Institute of Living in Hartford, the Newington Home and Hospital for Crippled Children, the William W. Backus Hospital in Norwieh, and the J. J. McCook Memorial Hospital in Hartford.

Dr. Tovell was espeeially recognized for his work in regional anesthesia. In 1939 he received the degree of Master of Seience in anesthesiology from the University of Minnesota. The title of his dissertation was "Anesthesia for Gynecologic and Obstetrie Procedures." He was one of the founders of the Amerieau Board of Anesthesiology, Inc., in 1937, and he was certified as a specialist in anesthesiology by this board in 1938. He knew the literature on anesthesiology well; he himself was the author of some 130 papers contributed to the literature of his speeialty and was associate editor of *Anesthesiology* from 1940 to 1955 and editor from 1955 to 1958. He was president of the American Soeiety of Anesthesiologists, Inc., in 1941; of the Amerieau Board of Anesthesiology, Inc., in 1947; and of the Aeademy of Anesthesiology in 1953. In September 1951 he was the Frederie Hewitt Leeturer at the meeting of the Royal College of Surgeons of England in London. He also gave an annual leeture on anesthesiology at the Yale University School of Medicine.

Dr. Tovell was called to aetive service in the United States Army August 26, 1942, with the grade of lieutenant colonel in the Medieal Corps. On September 24 he was assigned to the Eastern Theatre of Operations in Europe as senior consultant in anesthesiology; he was promoted to colonel in 1945 and released to civil life on Oet. 15, 1945. In 1945 he was awarded the Bronze Star for his work in developing a resuscitator and an ether vaporizer for use in the Eastern Theatre of Operations. The same year he reeeived the Médaille de la Reconnaissanee Franeaise from the French government and the Legion of Merit medal for his superior work as a medical officer.

Dr. Tovell was chairman of the Joint Committee on Anesthesia Hazards in 1941 and a consultant in anesthesiology to the Department of Defense in 1951. He was a member of the Subcommittee on Anesthesiology of the National Research Counеil from 1954 to 1956. In 1946 he was appointed a consultant in anesthesiology to the Veterans Administration, and from 1950 to 1953 he was chairman of the Board of Chief

Consultants of the Veterans Administration. From 1951 to 1954 he was chairman of the Armed Forces Service Committee of the American Society of Anesthesiologists, Inc.

Dr. Tovell was made an honorary fellow of the Association of Anaesthetists of Great Britain and Ireland in 1943, an honorary member of the Section of Anaesthesiology of the Royal Society of Medicine of England in 1944, and a foreign fellow of the Royal College of Surgeons of England in 1951. He was a fellow of the American Association for the Advancement of Science and a member of the American Medical Association, American Society of Anesthesiologists, Inc., International Anesthesia Research Society, Anesthetists' Travel Club, New England Society of Anesthesiologists, Connecticut State Medical Society, Hartford Medical Society, Hartford County Medical Association, Society of the Sigma Xi, and Alumni Association of the Mayo Graduate School of Medicine.

Dr. Tovell was married to Miss Lavina Ballantyne on July 6, 1929. He is survived by Mrs. Tovell and two children: Arnold Creighton Tovell, associated with the Beacon Press in Boston, and Mary Elizabeth Tovell, a registered nurse at Beth Israel Hospital in Boston.

JOHN S. LUNDY, M.D.

Book Reviews on Pain . . .

COMPREHENSIVE APPROACH TO THERAPY OF PAIN *by* A. LEWIS KOLODNY, M.D., *and* PATRICK T. MC LOUGHLIN, M.D., *145 pp., ill., Charles C Thomas, Springfield, Ill., 1966. $6.50*

This small book concerns itself with the daily problems of the physician who deals with pain. This book is not all inclusive, but deals, as far as the authors can, with the practical means and understanding of the basic principles of pain relief.

While this book is written from the viewpoint of the clinician rather than that of the pharmacologist, it nevertheless recognizes the importance of psychological mechanisms as well as pharmacologic, physiologic, and psychologic aspects of analgesics—and they are considered in some detail.

The book is divided into 12 chapters: "Historical Aspects of Pain Therapy," "The Pathways of Pain," "The Psyche, The Soma and the Pain Stimulus," "Analgesia," "Development of an Analgesic Drug," "Drug Addiction," "Non-Narcotic Analgesic Drugs and Their Actions," "Narcotic Analgesic Agents and Their Actions," "Analgesic Adjuncts," "Methotrimeprazine, a Phenothiazine with Analgesic Properties," "Misuse of Analgesic Agents," and "Comprehensive Approach to Therapy of Pain." There is a good index.

This book is very suitable for its purpose. Many drugs are considered and the presentation of side-effects is an important addition. This chapter has an adequate bibliography.

The problem of dealing with chronic pain is becoming increasingly important and we now have more treatises on the subject. In the course of time it may come about that many physicians will specialize in this most challenging field of practice.

This book is important to all physicians and they would do well to read it.

JOHN S. LUNDY, M.D.

CIBA FOUNDATION SYMPOSIUM: TOUCH, HEAT AND PAIN, *edited by* A. V. S. DE REUCK, M.SC., D.L.C., A.R.C.S., *and* JULIE KNIGHT, B.A., *389 pp., ill., Little, Brown & Co., Boston, 1966. $14*

This book on fundamentals concerning its subject is best described by its editors on the fly leaf of the book as follows: "In this second Ciba Foundation Symposium in a three-part discussion of the sensory function, twenty-five international authorities highlight current knowledge of the anatomical structures, neural processes, and biophysical principles underlying the sensations of touch, heat, and pain. In an attempt to prove the dynamic operating characteristics of the sensory systems, the papers and discussions present experimental and hypothetical material concerning stimulation from the external receptors to the brain, with emphasis on cell input-output relation in the skin, muscle, joint, and nerve fibres.

"The symposium members expand the psychophysical law that equal stimulus ratios produce equal sensation ratios, and present the results of years of laboratory experimentation to confirm the 'coupling of transfer function.' They suggest that in the study of pain, psychophysics provides one of the few realistic approaches.

"Specialists in the field of anatomy, cell biology, neurology, pharmacology, physiology, psychophysics, and zoology combine efforts to produce a valuable medical and chemical look at the macromolecular complexes within a membrane and show the relationship between the nerve cells of the peripheral and central nervous system."

This relatively small book includes a subject and author index. It seems adequately illustrated and outlined. There is a membership roll. We should all be greatful to the Ciba Foundation for adding to our storehouse of knowledge about the nervous system.

JOHN S. LUNDY, M.D.

Pregnancy in Patients with Osteogenesis Imperfecta

A Case Report and a Review of the Literature

CHARLES DUNHAM, B.S., and WILLIAM SPELLACY, M.D.
Minneapolis

Osteogenesis imperfecta (O.I.) is a rare inherited disorder of the maturation of mesenchymal tissue resulting in a syndrome of complications. These include fractures, blue sclerae, deafness due to otosclerosis, hyperflexibility of the joints, and poorly formed teeth (odontogenesis imperfecta).[1–4] The disease is thought to be transmitted through an autosomal dominant gene resulting in the passage from 1 affected parent to 50% of the offspring.[5,6] The genetics are complicated by incomplete penetrance and a variable latency period.[5,6]

The presence of this condition in a pregnant patient presents the physician with several problems. The maternal problems include dystocia due to pelvic contraction from previous fractures, the production of maternal fractures at the time of delivery, and respiratory insufficiency due to chest cage deformities. The problem of the afflicted infant is the production of fractures during delivery.

The purpose of this paper is to present a review of the literature of pregnancy in patients with osteogenesis imperfecta and to report 2 additional cases.

CASE HISTORIES

Case 1. A 27-year-old primigravida was seen for the first time on Dec. 23, 1948, in active labor and with the cervix completely dilated. She had had no prenatal care. Her menarche was at age 15 and her menses had been very irregular with periods of amenorrhea from two to nine months and menstrual flows lasting from seven to ten days. She had a history of multiple fractures and was noted to have blue sclerae. X-rays of the skull, chest, pelvis, and long bones disclosed bony changes consistent with a diagnosis of O.I. The pelvic examination showed a generally contracted pelvis. Her laboratory studies showed a normal complete blood count, urinalysis, calcium, phosphorus, blood urea nitrogen, and serology.

The fetal head was felt to be soft and easily indented. On Dec. 24 the patient spontaneously delivered a 2,690-gm. female infant over a left mediolateral episiotomy using pudenal block anesthesia. On physical examination the infant was noted to have extreme thinness of the skull, lethargy, and normal colored selera. X-rays of the infant taken on Dec. 27 disclosed a fracture of the left clavicle and numerous anomalous lines in the skull interpreted as possible old fractures. The infant has subsequently incurred fractures of the right humerus at 4 months of age and of the left femur at 7 months. A diagnosis of O.I. was made but there have been no further follow-up examinations.

Case 2. A 31-year-old primigravida had an estimated date of confinement of Oct. 30, 1965. The patient had her menarche at age 14 and had regular menstrual periods every twenty-eight days for 4 days' duration. O.I. had been diagnosed at birth. Multiple fractures of the extremities had resulted in shortening and severe deformities of her legs. She had undergone many orthopedic procedures and had also had bilateral myringotomies for the correction of a hearing defect. The physical examination showed a 122-lb. deformed female with blue sclerae and multiple dental caries. Her blood pressure was 130/88 mm. Hg. X-ray pelvimetry showed a deformed contracted pelvis with a transverse diameter of the midplane of 8.4 cm. The patient was ad-

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mitted to the hospital on Oct. 7 because of a blood pressure of 144/110 mm. Hg. Her blood urea nitrogen and urinalysis were normal. Her vital capacity was 1.8 liters in 1 second and a total of 2.1 liters. Her blood type was O cDe and she had a normal electrocardiogram, complete blood count, serology, Pap smear, glucose tolerance test, and serum electrolytes. A fetogram on Oct. 14 showed no radiographic evidence of O.I. in the fetus. The blood pressure was controlled medically. On Oct. 26 the patient went into spontaneous labor and an elective low segment, transverse cesarean section was performed. A 3,540-gm. normal female infant was delivered. The postpartum course for the patient and her infant was normal. The infant was noted to have blue sclerae but X-rays have shown normal bone development.

DISCUSSION

Osteogenesis imperfecta is a rare medical problem; however, a large number of cases have been reported. The lack of reports of pregnancy in these series suggests either a decreased fertility rate or a decreased opportunity for the patients to become pregnant. There is no adequate data given concerning ovulation.

There have been 8 reported cases of pregnancy in patients with O.I. in the English literature. A review of these is seen in the table. All of the reported patients had a history of fractures, and blue sclerae were noted in 6. Other manifestations include scoliosis and odontogenesis imperfecta. Seven of the patients were noted to have pelvic abnormalities. One patient was aborted at sixteen weeks because of potential complications of respiratory insufficiency.[3] Seven patients were delivered vaginally and 1 delivery resulted in the fracture of both right maternal pubic rami.[7] In 1 woman an indirect complication occurred when at thirty-eight weeks she had spontaneous rupture of membranes, slipped on the amniotic fluid, fell to the floor, and fractured her right hip and pelvis.[8]

The obstetrical management of these cases requires a knowledge of the potential problems. The first decision must be related to the mode of delivery. A maternal indication for cesarean section may be the bony pelvic abnormalities while a fetal indication is the possibility of induced fractures from traumatic delivery.

In order to obtain more data concerning the infants, one must consider those patients who have acquired the disease through spontaneous mutation, thus being born of normal parents. There are many such reports available. These include several reported where O.I. was diagnosed in utero by X-ray.[9–12] These reports show that fractures can occur before delivery and thus, vaginal delivery could be disasterous. The in-

SUMMARY OF PREGNANCIES REPORTED IN PATIENTS WITH OSTEOGENESIS IMPERFECTA

Author and date	*Patients' age*	*Maternal disease manifestations: Fractures*	*Blue sclerae*	*Deafness*	*Maternal pelvis*	*Method of delivery*	*Gestation or birth weight, or both*	*Infant outcome*	*Complications of delivery*
Graffee[13] 1953	32	+	+	NR*	Marked Asymmetry	Vaginal	2,370 gm.	Soft skull Blue sclerae old fractures	None
Staples[7] 1954	18	+	+	NR*	Normal	Vertex Low Forceps	3,147 gm. 40 weeks	Blue sclerae No fractures	Fracture, maternal pelvis
	23	+	+	NR*	Normal	Vertex Outlet Forceps	3,147 gm. 40 weeks	Blue sclerae No fractures	None
Johnson[14] 1957	28	+	+	NR*	Gynecoid adequate	NR*	3,320 gm.	Blue sclerae No fractures	None
						Frank breech extraction	3,630 gm. 42 weeks	Blue sclerae No fractures	None
Cohn[8] 1962	27	+	+	NR*	Fresh fracture 2° to fall	Vertex Low Forceps	38 weeks	Normal	None
White[15] 1963	23	+	+	No	Prominent spines; small outlet; hooked sacrum	Vaginal	3,820 gm. 40 weeks	Normal	None
Bender[3] 1965	34	+	–	NR*	Very small	Induced abortion	16 weeks	Spina bifida Rachischisis	None
Evans[2] 1966	23	+	NR*	NR*	Gross deformity	Classical C-section	34 weeks 1,675 gm.	Normal	None
UMH-1 1948	27	+	+	–	General contracted	Vertex Vaginal	2,690 gm.	Soft skull, fracture left clavicle	None
UMH-2 1965	31	+	+	+	Deformed and contracted 8.4 cm. interspinous	Low segment cesarean section	3,540 gm. 40 weeks	Normal	None

*NR indicates not reported

fants with severe O.I. are often stillborn or die in the neonatal period. The cause of death is rarely given but severe trauma may be presumed to be important. It is therefore suggested that liberalization of the indications for cesarean section in these cases may be beneficial.

During delivery the physician must minimize the trauma to the patient's legs and pelvis realizing that instrumentation, positioning, or vaginal passage of the fetus may result in fractures.

Finally, the physician must be aware of the possible decreased respiratory reserve. If there is doubt, tests of pulmonary function before delivery would be valuable.

SUMMARY

Two cases of maternal O.I. have been reported and 8 cases from the literature reviewed. The likely complications have been discussed. Many of the affected infants can be diagnosed in utero. It is hoped that by liberalization of the use of cesarean section in these cases improvement in both maternal and fetal outcomes may occur.

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Continued from page 282

ACUTE BRONCHIOLITIS AS A CAUSE OF CHRONIC OBSTRUCTIVE LUNG DISEASE IN ADULTS

David E. Dines, M.D.

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A. MALCOLM CAMERON	Minot
J. D. CRAVEN	Williston

REFERENCE COMMITTEES

1. *To consider Report of President, Secretary, Executive Secretary and Treasurer:*

ROBERT E. HANKINS, Chairman	Mott
ROY E. KULLAND, Vice-Chairman	West Fargo
C. ROBERT MILLER	Bottineau
WM. T. POWERS	Grand Forks
N. F. GOLTZ	Fargo
WILLIAM RIECKE	Bismarck

2. *To consider Reports of Council, Councillors, and the Report of the Representative of the Blue Shield Board of Directors:*

G. L. COUNTRYMAN, Chairman	Grafton
R. D. McBANE, Vice-Chairman	Devils Lake
HENRY A. NORUM	Fargo
B. F. AMIDON	Fargo
O. V. LINDELOW	Bismarck

3. *To consider Report of Delegate to the A.M.A., Medical Center Advisory Council, the Committee on Medical Education and Special Committees:*

J. V. MILES, JR., Chairman	Jamestown
R. J. ULMER, Vice-Chairman	Fargo
H. W. EVANS	Grand Forks
A. MALCOLM CAMERON	Minot
M. J. E. JOHNSON	Bismarck

4. *To consider Reports of Standing Committees, except Committee on Medical Education and Committee on Medical Economics:*

M. A. K. LOMMEN, Chairman	Bismarck
M. H. POINDEXTER, JR., Vice-Chairman	Fargo
B. J. CLAYBURGH, Alternate	Grand Forks
JOHN GOVEN	Valley City
W. B. HUNTLEY, Alternate	Minot
KENNETH WAKEFIELD, Alternate	Cooperstown

5. *To consider Reports of the Committee on Medical Economics, The North Dakota Advisory for Hospital and Related Construction and The Ad Hoc Committee on Heart Disease, Cancer and Stroke:*

A. C. BURT, Chairman	Fargo
J. J. MOSES, Vice-Chairman	Bismarck
G. H. HILTS	Cando
W. W. NELSON, Alternate	Grand Forks
D. E. SKJEI	Williston
G. R. RICHARDSON	Minot

6. *Committee on Resolutions to Include New Business:*

E. G. VINJE, Chairman	Hazen
E. O. HIEB, Vice-Chairman	Jamestown
L. A. GILTNER	Minot
J. A. SANDMEYER	Grand Forks
PAUL BEITHON	Wahpeton
W. C. HANEWALD	Dickinson
RALPH DUNNIGAN	Bismarck

7. *Committee on Credentials:*

R. J. ULMER, Chairman	Fargo
(Also Serves on Reference Committee No. 3)	
D. E. SKJEI, Vice-Chairman	Williston
(Also serves on Reference Committee No. 5)	

PROCEEDINGS: HOUSE OF DELEGATES EIGHTIETH ANNUAL MEETING OF THE NORTH DAKOTA STATE MEDICAL ASSOCIATION

May 5, 1967 — Bismarck, North Dakota

FIRST SESSION

The first session of the House of Delegates of the North Dakota State Medical Association was called to order by the Speaker of the House, Dr. P. O. Dahl, at 8:00 a.m. in the Prince Hotel, Bismarck, N. D.

Dr. Ulmer, Chairman of the Committee on Credentials reported that all credentials were in order and that there was a quorum present. He also reported that Dr. B. J. Clayburgh, Alternate, would be seated in the House for Dr. R. H. Leigh, the regular delegate; Dr. W. W. Nelson, Alternate, would be seated in place of Dr. K. S. Helenbolt; Dr. N. F. Goltz, Alternate, for Dr. Jaehning; Dr. W. B. Huntley, Alternate, for Dr. B. Jayapathy; and Dr. Kenneth Wakefield, Alternate, for Dr. James Little.

Secretary Buckingham called the roll and the following doctors responded:

Doctors: H. A. Norum, Fargo; M. H. Poindexter, Fargo; A. C. Burt, Fargo; Roy Kulland, West Fargo; B. F. Amidon, Fargo; R. J. Ulmer, Fargo; Paul Beithon, Wahpeton; N. F. Goltz, Alternate, Fargo; G. H. Hilts, Cando; R. D. McBane, Devils Lake; C. R. Miller, Bottineau; J. A. Sandmeyer, Grand Forks; G. L. Countryman, Grafton; H. K. Evans, Grand Forks; W. T. Powers, Grand Forks; W. W. Nelson, Alternate, Grand Forks; B. J. Clayburgh, Alternate, Grand Forks; L. A. Giltner, Minot; G. R. Richardson, Minot; A. M. Cameron, Minot; W. B. Huntley, Alternate, Minot; J. W. Goven, Valley City; O. V. Lindelow, Bismarck; J. J. Moses, Bismarck; M. A. K. Lommen, Bismarck; R. J. Dunnigan, Bismarck; Edmund Vinje, Hazen; W. C. Riecke, Bismarck; E. O. Hieb, Jamestown; J. V. Miles, Jamestown; D. E. Skjei, Williston; R. E. Hankins, Mott; Kenneth Wakefield, Alternate, Cooperstown.

There were 28 delegates and 5 alternate delegates, duly authorized and present.

The following also attended the House of Delegates meeting: Drs. R. C. Painter, G. W. Toomey, V. J. Fischer, R. W. McLean, Dean Strinden, Mr. Lyle Limond and Mr. W. David Coyner.

The motion was made by Dr. Evans, seconded by Dr. Giltner, that the reading of the minutes of the last meeting in Fargo be dispensed with and that they be approved as printed in the Journal-Lancet. Motion passed.

The motion was made by Dr. Evans, seconded by Dr. Giltner, that the reading of the minutes of the Interim Meeting in Fargo, December 1966 be dispensed with and that they be approved as printed in the Handbook. Motion passed.

Dr. Dahl introduced the new Field Representative from the American Medical Association, Mr. W. David Coyner, who is replacing Mr. Sherwood Williams, now Dean of the Medical School in Saigon. Mr. Coyner was formerly with the Florida Medical Association and offered his services at any time.

Dr. Richard Prouty, State Toxicologist, was next introduced by Speaker Dahl.

Dr. Prouty: I will not take up much of your time, but I do want to personally make you aware of a piece of legislation that was passed in the last legislative session that affects all of you as a physician; namely, Senate Bill 44 which provides for a traffic fatality study and beginning the first of July of this year and for the next two years, through July 1969, we will be required to collect blood samples from all fatally injured traffic victims in the state; not only drivers but passengers and pedestrians. I have sent a letter of instructions to all of the medical groups throughout the state; physicians, hospital administrators, clinics and coroners. But, I asked if I could appear before you so that you may carry back to your districts a plea for your cooperation in this program.

I do not intend to go through the text of the law. I can advise you that the law is so written that the collectors of these blood samples shall be immune from all liability, civil or criminal litigations.

The reason for this legislation is to determine the role of drugs and alcohol in traffic accidents in the state. The State of Illinois has had this and the results were quite

illuminating as to the role that alcohol played in traffic accidents. I have some pamphlets that each of you may pick up to take with you. If any one of you have any questions, I will be glad to furnish the answers. Kits will be furnished from my laboratory to clinics, doctors and coroners for collecting these blood specimens, and these will be sent to the laboratory. I am making a plea that the physicians help us in collecting these bloods. We want to get these specimens 24 hours after death, and it is mandatory that you get this to the state laboratory immediately.

Dr. Wakefield: Can the findings of these blood specimens be used in any civil or criminal actions?

Dr. Prouty: No. These are to be confidential. The results of any particular analysis can not be used for any civil or criminal procedures.

Dr. Hilts: Are these kits going to be sent out, or is this by request?

Dr. Prouty: I will send these out to each of the hospitals, clinics, coroners and private doctors.

Dr. Powers: How long after the accident is this to be acquired?

Dr. Prouty: After they die, within 24 hours.

Dr. Miller: If they have been dead for a day or two, and they have just been found, then just what procedure do you follow?

Dr. Prouty: Draw this anyway, but get the heart blood. In the instructions in this pamphlet, I have made an attempt to give suggestive procedures for drawing these bloods. One of the problems that comes up in post-mortem bloods is that drawn from the heart. I have suggested wherever possible, that the blood be collected from a large peripheral vessel at some point distal to the heart, preferably the femoral or subclavian or external jugular veins. If this is not possible, then in drawing blood from the heart, insert the needle through the fourth intercostal space immediately to the left of the sternum.

Incidentally I wrote the Attorney General for an opinion, because this has come up in Fargo where a licensed embalmer is qualified to draw these bloods, and they are well qualified to draw these bloods. I realize that accidents are very often going to take place some distance from a doctor. Normally, they are going to have to open one of the vessels and I wanted to get this clear as to whether or not they are qualified to draw this blood.

The blood specimen must always be collected from the body of the decedent, prior to embalming, because of alcohol or drug content. This study is greatly needed in order to provide data in regard to the state's program of traffic safety. We want to find out actually what is happening here in our state.

Dr. Vinje: How about an accident case in which the person might last longer than 24 hours?

Dr. Prouty: If they die within 24 hours of the accident, even though they are admitted as a patient, the blood should be drawn post-mortem. Regarding the question of the seriously injured; they are not covered under this law. If they agree, and sign a statement, it will be all right to draw this blood, if we could get this. This is strictly fatalities.

Dr. Dahl: Thank you very much for coming and speaking before the House, Dr. Prouty.

I wish to say a word about the reason for starting so early this morning. I discussed this with our Executive Secretary and the staff, because last year at Fargo there was too short a time between the session of the House and the Scientific program. The program starts at 10:30 a.m. and in our city this is held in the World War Memorial building, three blocks from here. We do want to be there on time. The schedule of the House is a very busy one so we felt that by starting early we could be out of here promptly at 10:00 or 10:20. You may rest assured that we will be out of here so that you may reach the Auditorium for the first Scientific lecture.

At this time, I would like to say a word regarding Mr. Limond. I do not know whether you have all had a chance to greet him this morning. He has been in the hospital since October and will still be in the hospital after this meeting. His physicians allowed him to attend these meetings here in the hotel. Lyle, I speak for every member of the House; we are certainly happy you are here. We can hardly wait until you are going full blast.

(Round of applause for Mr. Lyle Limond).

Our State Association has been fortunate in having a great many fine presidents. The past year has been no exception. We have been blessed with a very outstanding man who has given us a great deal of time. It gives me great pleasure at this time to introduce our State President, Dr. Glenn W. Toomey.

Dr. Toomey: After putting up with me for a year, such a claim is flattering in the superlative. Phil asked me if I have an address but I do not have. Out of deferment to the incoming President, I have no prepared remarks this morning. I have said it all in the handbook. I do not have much more to say. The high-light of this meeting is seeing Lyle back. I want to tell you that I think I make a very good valet.

I was going to say something about committees and their functions. However, there are a great many committees and they have all done outstanding work. Dr. Foster and his committee especially have done a beautiful job on the revision of the Constitution and By-Laws.

I told our incoming President, Dr. Fischer, that I was going to bring this up this morning. I think in the future if you would have the president of your District Society invite the State President for a visit, it would save some embarrassment on the part of the State President. I did not make a visit to one of our district societies, because I was not invited to do so, and I did not have the nerve to call them and ask them if they wanted me. The state president is in a frustrating position at times, as I stated in the handbook. One has a title but no power and the president receives a good deal of criticism and blame for things that are done and not done. I want to thank all of you for your help and for working on committees. I thank you for the privilege and honor of serving you for the past year. I only ask that you do not send me to the "glue factory" entirely, as I would like to carry on somewhere in the Association.

Dr. Dahl: Thank you, Dr. Toomey. You know, deep in your heart, you are going to have plenty of jobs to do.

It is always one of the highlights of the meeting of the House to have the opportunity to hear the man who is going to command our Association for the ensuing year. It gives me great pleasure at this time to introduce the President-Elect, Dr. V. J. Fischer of Minot, for his Inaugural Address.

INAUGURAL ADDRESS

Dr. Fischer: We are convened for the Eightieth Annual Meeting of the North Dakota State Medical Association. I wish to thank this organization with a great sense of humility for elevating me to this position. I stand today in the shadow of leaders who have gone before, who have been an inspiration to all of us and have gone on demonstrating their leadership and abilities. I shall follow the path that has been shown to us to the best of my ability and shall try in all sincerity to merit your trust.

This past year has been a year of great milestones in the history of medicine. There has been the advent of Medicare and

the implementation of Medicaide. The State of North Dakota has been fortunate in this advent by having the efficient home organizations with previous experience in this field; namely, the North Dakota Physicians Service and Blue Cross as carriers in the first program. The tireless efforts of these organizations for the benefit of the patients have prevented, so far at least, the milestone of medicine from becoming the millstone some had prophesied, and which still could occur if our social minded friends and legislators who don't wait to catch their breath and let the present program seek its level of utilization; only then can the good, the cost and the inefficiencies be properly assayed so corrections can be made. We are all aware that a new program is never without fault; however, such faults should be corrected equitably and not be compounded by new and more expanded experiments before the first is corrected. Medicare is now the law of the land and only because of the tremendous cooperation of you physicians has the program been able to operate. I think you are all to be commended on your professional spirit and sense of obligation that has made this program operate as well as it has. Certainly many people have been aided and others, through the confusion of implementation, have been hurt. It is by your continuing effort that any deficiencies will be corrected and I know no patient will ever suffer through you because of governmental blundering.

Let us turn our attention to another facet of this age.

This present age is being known as the age of mass production and mass effort. This mass effort has fitted into the medicine in various ways. More and more we are confronted with patients whose problems and anxieties are the result of these mass programs. The patient who is fighting a struggle to be an individual and is finding himself slowly but surely losing the fight. He is developing an uneasy sense of emptiness and frustration. He is filling a slot in which he has less and less control. His life is planned for him and he must drift along this river of anonymity or stand like a rock in midstream producing the rapids and turbulence of individual thought and questioning. It is because physicians are not content to just go along in the conveyer belt of anonymity but have stood up as boulders in midstream to question and to analyze that they have reaped the adverse publicity and the headlines of newspapers as obstructionists. You have dared to question the dictates of the planners and politicians. You have received criticism, seldom however, in its true and honest light. Basically you are better educated, have a finer insight through constant training and keen observation than other professions with few exceptions. You are individuals, all chiefs, and here lies one of the problems. As chiefs, many find it difficult to work together to form a strong central organization. This includes the District Medical Association, the State Medical Association, and the American Medical Association. I know that some of the physicians are not joining the AMA because of some of its actions, hut do they know all the work it has done in the past and is doing every day of which they are not aware? When one of us fails to see the need for a central organization to implement our purposes, then he is knocking his own purposes as an individual. Many do not realize that as an individual physician they can be heard at the AMA in the reference committees' sectional meetings. Where can a greater democracy than this be obtained in such a large organization? It is one of the few great organizations where an individual from the grass roots can be heard.

Now let us ask a question. What can we do if we work together? This simple question is a reminder of the irony of the present state of mankind. It shows our persistent struggle between wisdom and foolishness, our strength and our weaknesses, our creativeness and our idealism versus our self-destruction and baseness. It reminds us of the strange fact that even now with our enlarged range of knowledge, men all over are passively continuing to accept policies and programs of all kinds that are frittering away their rights and substance and are generally nibbling away their existence.

Some say this is human nature. Is this the real reason for our inability to cope with our problems? No, it is usually greed, prejudice, envy, fear, misunderstanding and arrogance. Perhaps we think more of our differences than of our opportunities. Perhaps we are still in the state of mind where traditional attitudes born of past wants and conflicts influence us more than the opportunities of the present. We must gain enough confidence in ourselves and in each other to turn our ideas and our potential into reality. It is a historic fact that when men have worked together in enthusiasm and loyalty to a commonly held idea, the end results have benefited all succeeding ages.

There are two facets in which I should like to see our State Medical Association work toward in the next year. There are many more, but these are two prime needs which we should be looking into and studying. First, I feel that hospital planning, long range, regional, local and state is an important part of our effort. The Heart, Cancer, Stroke Program of the Federal Government is only a small portion of what our planning should include. The Ad Hoc Committee could probably be used as a nucleus for this study. Secondly, I feel that we should be instrumental in establishing medical society review committees. It is important that these review committees he on the basis of our medical society and not on the basis of insurance carriers. Guidelines for establishing medical society review committees has been re-established and reaffirmed by the A.M.A.'s House of Delegates, (J.A.M.A. 194:1021, Nov. 29, 1965) and four additional principles have been adopted:

The medical profession should assume leadership in establishing forums through which the judgment, training and experience of physicians in practice in the community can be brought to bear in the review of questionable cases involving either: (1) usual customary or reasonable charges; (2) the effectiveness of hospital use, or (3) the necessity of the services rendered.

Medical societies should establish and support professional review committees and provide guidance and advice required to assure their optimal function.

To assure consistency in the judgment values applied and equitable decisions for all segments of the public, the same committee should be used for consideration of cases in its area of responsibility regardless of source of submission.

To achieve maximum effect of the committee's educational potential, representations of health insurance and prepayment plans should be available to the review committees and their participation should be sought as consultants.

I do not believe that insurance carriers including the "Blues" should ever be put in the position of dictating to doctors or hospitals how medicine is to be practiced. In order to arrive at a just settlement, it should be possible by anyone concerned to submit controversial claims to a local review committee. Here any of the principles mentioned at the onset could be discussed and properly adjudicated.

In the present state of third party carriers, if we the Medical Society will not set up this type of organization and review committee, it shall he done for us. Let it not be said that we physicians have missed the opportunity to guide the course of medicine. We must be leaders not obstructionists.

This past year it has been my privilege to attend the sessions of the AMA in Chicago, the interim meeting in Las Vegas, the North Central Medical Conference in Minneapolis and the meeting of our sister state to the west, the Montana State Medical Association at Bozeman. As always new enthusiasm and new sources of inspiration are received at these meetings. We are now meeting in our own state association and it is your turn to stand up and to be heard. This is your association—work for it and in it! I am sure this association can accomplish many things and with the loyalty of our members, our executives, and hopefully with the continuing help of our helpful and loyal secretary, Mr. Limond, this association will find this coming year a fruitful one.

Thank you again for this honor and your confidence in me.

Dr. Dahl thanked Dr. Fischer for his inspiring talk and added that he hoped Dr. Fischer would find that the House of Delegates is cooperative in the areas mentioned.

Dr. Dahl next referred all the reports printed in the Handbook to the proper reference committees for their action.

REPORT OF THE PRESIDENT

The activities of the State Association will be detailed in the various reports of the Handbook. It has been a pleasure and an honor to serve the North Dakota State Medical Association during the past year. As you know, we have had many problems, some of which have been solved and some are unsolved, many are still in a state of flux. The office of President is most frustrating at times. One has a title but no power—which perhaps is well—but at the same time, the President receives a good deal of criticism and blame for things that are done and not done.

During the past year, the President attended most of the major meetings, excepting the Interim AMA meeting in Las Vegas. I attended the AMA meeting in Philadelphia; the AMPAC meeting in Washington, D.C.,—which was most instructive; the AMA meeting in Chicago in June; the Public Relations meeting of the AMA; the meeting of the State Presidents in Chicago in August; and the North-Central Conference in November. These meetings were all most enlightening; however, no one

person can begin to absorb all of the ideas or problems which are brought up and discussed. If I were to pick the meeting of most value, I would pick the AMPAC meeting in Washington, which actually was a workshop and would be a good meeting for more of our members to attend. It was most instructive and informative.

Our North Central Medical Conference is becoming stronger and more functional as it reaches maturity. Being that we belong to a group of states who singly have very little power, it is certainly a good medium for us to gain recognition in cooperation with Wisconsin, Nebraska, Minnesota, South Dakota and Iowa as our conglomerate vote means something at the national meetings. I am happy to say that this year our own Clifford Peters of Bismarck is President-Elect of the North Central Medical Conference and I hope he will receive enthusiastic support from members of this Society.

The American Medical Association still receives a great deal of criticism from members of our society who, I'm sure, have never attended a meeting of the AMA and who really do not understand its functions and objectives. It has been proposed to me, and naturally it is up to the Society to decide, that each year the North Dakota Society—in addition to its delegate and alternate delegate—send one man from one of the Districts in a rotating manner to the spring AMA meeting, so we would have three men there every year. A man from each District, within a period of 10 years, would have attended an AMA meeting along with the delegate and the alternate. This could be any member elected by the local society or possibly the Councillor. This maybe would have some value in getting the men in various Districts better acquainted with the AMA.

Some of our committees have been extremely active this year, as you well know. I especially wish to mention the work of Dr. Victor Lindelow of Bismarck and compliment him on the fine job that he has done with the Medical Economics Committee. As you all know, he fought for, and eventually obtained, the usual and customary fee in the face of a good deal of opposition. Dr. Lindelow and his Committee are to be complimented. As you know by now, the Committee on Legislation did an excellent job during the session of the Legislature. You know that we obtained most of our objectives without too much trouble and credit must go to the Committee, and especially to Dr. William Buckingham of Bismarck who gave freely of his own time and efforts as our lobbyist.

I have had the pleasure of visiting all except one or two of the local Societies during the year. I have been most impressed by their scientific meetings and found it very stimulating to get around the state and see that we all have the same problems that are confronting us and which will confront us in the future. We all know that we are not through with medical legislation by any means. In fact, it is just beginning. However, through our local Societies and our state Societies, we can present a united front to at least hold the line and fight for our rights as the problems arise. One of the satisfying experiences of serving as President is the privilege of meeting with these local Societies and becoming better acquainted with my own confreres in the state. I would urge some of the Societies to have more meetings during the year and I would urge all of the Societies to encourage their younger men to become active, not only locally, but in the State Society; to attend the meetings; and offer their services on the various committees.

After having attended local Society meetings and national meetings during the past year, I am very proud to say that I live and practice medicine in North Dakota. I think we can all be proud of the standards of medicine in this state. We can be proud of our integrity. When I listened to some of the questions brought up at the Public Relations meeting in Chicago, I was amazed, and realized just how clean we must be out here. I feel that if every state and community worked under the same standards of ethics as men in North Dakota, the recent publication, "The Healers" would never have been written.

We were most unfortunate in losing the complete services of our Executive Secretary, Lyle Limond, beginning in November of 1966. However, Lyle has done an excellent job of direction from his hospital bed, not only of our office in Bismarck, but he was also well able to keep in touch with the proceedings in the Legislature. With the very efficient help from Mrs. Margaret Fremming and Mrs. Myrtle Kohler, we have been able to carry on very well. I wish to thank the staff of the Bismarck office for their dedicated efforts and I want to state that I did miss Lyle Limond's company and counsel in my visitations around the state following his accident. I wish him an accelerated recovery.

If I have neglected expressing special thanks to any of the members or committees who have done special or outstanding work, it is certainly not by intention but by oversight.

In closing, I wish to thank you for electing me President of your association last year. I can honestly say that I do not feel I have accomplished an awful lot; however, it has been a privilege and a pleasure to have represented you at State and National meetings and I am grateful for the opportunity.

Respectfully submitted,
(Signed) G. W. Toomey, M.D., *President*
North Dakota State
Medical Association

SECRETARY'S REPORT

The following report of the secretary is respectfully submitted:

Membership: The total membership for 1966 was 509. Of this number, 460 paid the regular membership fee, 3 paid ½ year's dues, 3 paid ¼ year's dues, 11 were on a retired basis, 4 on a limited basis, 2 associate members and 26 were honorary. Nine physicians passed away during the year and several have left the state. New members, however, are being steadily added to our roster.

Table 1 shows the annual membership for the past five years.

TABLE 1
COMPARISON OF ANNUAL MEMBERSHIP

	1962	1963	1964	1965	1966
Paid Memberships	436	449	455	459	466
Honorary Memberships	25	21	27	24	26
Limited and Retired	4	6	8	12	15
Associate Memberships	-	-	-	-	2
Dues Cancelled (age exemption, illness, military service)	1	1	1	-	-
	466	477	491	495	509

Table 2 shows the annual dues payments according to the cut-off date for preparation of this report for the past five years. The cut-off date of April 11, 1967 has given members a grace period of six weeks in which to pay their current dues, even though the Constitution and By-Laws of the Association states that all assessments must be forwarded to the Secretary not later than the 1st day of March of each year. You will note that

even with the extension of time and despite the fact that members were urged to make dues payments as early as December, quite a large number of members are delinquent in dues payments.

TABLE 2

	April 12 1963	April 3 1964	April 2 1965	April 4 1966	April 11 1967
Paid-up Members	388	412	412	415	386
Honorary Members	21	25	22	26	29
To be Honorary	1	1	3	5	2
Dues Cancelled	1	1	-	-	-
Associate	-	-	1	2	-
Limited	2	3	2	2	4
Retired	2	2	5	8	6
Regular ½ Yr. 1966	-	-	-	-	1
Regular ¼ Yr. 1966	-	-	-	-	1
Ltd., 1964, 1965, 1966	-	-	-	-	1
	415	444	445	458	430

STATE ASSOCIATION MEMBERSHIPS

1966

Dist.	Reg.	Rtd.	Ltd.	Assoc.	Hon.
First	103	7	1	–	5
Second	38	–	–	–	2
Third	78	2	–	–	4
Fourth	60	–	1	–	4
Fifth	11	–	–	–	1
Sixth	90	1	–	2	5
Seventh	30	1	2	–	1
Eighth	21	–	–	–	–
Ninth	25	–	–	–	4
Tenth	10	–	–	–	–
	466	11	4	2	26

1967

Reg.	Rtd.	Ltd.	Assoc.	Hon.
86	5	–	–	4
35	–	–	–	2
62	–	–	–	6
43	–	1	–	7
9	–	–	–	–
65	–	–	–	5
31	1	2	–	1
22	–	1	–	–
25	–	–	–	4
8	–	–	–	–
386	6	4	–	29

(Dues payments also made for ¼ year member, 1st Dist., '66 ½ year member, 6th Dist., '66 Ltd., 1964, 1965 and 1966, 8th Dist.)

A.M.A. GENERAL MEMBERSHIPS

	1966	April 11, 1967
First	111	94
Second	39	36
Third	82	67
Fourth	63	49
Fifth	12	9
Sixth	96	69
Seventh	33	34
Eighth	21	22
Ninth	27	27
Tenth	10	8
	494	415

Representation in the House of Delegates is based on one delegate for each 15 paid-up members (or major fraction thereof,) together with honorary members. Delegates from the local societies should be seated on this basis.

The continued cooperation on the part of all members in prompt payment of their dues will be greatly appreciated.

Respectfully submitted,

(Signed) W. M. Buckingham, M.D., *Secretary*
North Dakota State Medical Association

REPORT OF THE EXECUTIVE SECRETARY

GENERAL COMMENTS: Your Executive Secretary was able to attend quite a few state and national meetings before he fell at home and broke his hip last fall.

Mrs. Margaret Fremming and Mrs. Myrtle Kohler have carried on the work in the central office in a rather exemplary manner during my absence.

LEGISLATION: Your Executive Secretary missed the lobbying chores at the 40th session of the North Dakota Legislative Assembly. William Buckingham, M.D., of Bismarck, took over the job of lobbyist in a very exemplary manner. He is deserving of all our thanks for a "job well done."

The Association had two bills and one concurrent resolution introduced during the session. All were indefinitely postponed. H.B. 899 (sets standards to qualify for medical assistance care and services) and S.B. 227 (relates to contract of the Public Welfare Board with private agencies or carriers) were the two bills introduced. The resolution was Senate Concurrent Resolution KKK (directing the LRC to study the miscarriage and abortion statutes of the state to determine whether or not they are in need of amendment). The resolution was introduced by Senators Stafne, Ringsak and Longmire. The Association may wish to try and have introduced a bill dealing with abortion in the 1969 Session of the North Dakota Legislature.

We were fortunate in having a doctor of medicine in the Senate, William Stafne, M.D., of Fargo, plus a clinic manager. There were two clinic managers in the House.

Please note the report from the Committee on Legislation for the complete listing of legislation followed during the past session. The members of this Association who visited the Legislative Assembly to testify on bills and to contact their Senators and Representatives are to be commended for their efforts.

On the national scene, Senator Russell Long of Louisiana has introduced a bill (S.1303) in the new Congress which differs considerably from his "compulsory generic" prescribing bill (S.3614) in the 89th Congress. His latest measure would establish a national formulary and a price guide under which federal payment would be made to beneficiaries of health care programs under various titles of the Social Security Act. Hearings by the House Ways and Means Committee on the Administration's proposal on Social Security-H.R. 5710 were scheduled to end on April 6. There are many reasons why the further expansion of Medicare should be opposed. Basically, it is a bad law—extremely costly, difficult to administer, the degree of government control is not compatible to good medicine, and it taxes workers and employers to finance care for millions of people who are self-supporting.

PHYSICIAN'S PLACEMENT SERVICE: This office continued to give service to communities, individual physicians and/or medical groups seeking physicians.

U.N.D. MEDICAL SCHOOL SCHOLARSHIPS: The

1966 winners of the Association's Scholarship Prizes (totalling $500.00) offered at the School of Medicine were as follows:

Highest average mark in Anatomy—Daniel Moline, Rolette, N. D.

Highest average mark in Physiology-Pharmacology—Margaret Wright, Fargo, N. D.

Highest average mark in Microbiology—Margaret Wright, Fargo, N. D.

Highest average mark in Pathology—David Mersy, Grand Forks, N. D.

Highest average in the Work of the First Year—Lloyd K. Everson, Grafton, N. D.

CHAMPUS: Major additions to the Government's medical care program for the Uniformed Services (Military Medicare—Office For Dependents' Medical Care—ODMC) have been provided by legislation signed by the President on September 30, 1966.

Under the Military Medical Benefits Amendments of 1966 (Public Law 89-614), the Civilian Health and Medical Program of the Uniformed Services (CHAMPUS) provides both hospitalization and outpatient care in civilian facilities for dependents of active duty members, retired members entitled to retired, retainer, or equivalent pay (including retired reservists), their dependents, and survivors of deceased active duty and eligible retired members.

Also added is a program of care in civilian facilities for active duty dependents who are mentally retarded or physically handicapped.

Present space-available care of retirees and dependents in Uniformed Services facilities will continue and some new benefits have been added.

All members of this Association and clinic managers have been sent "Fact Sheets" describing this expanded program in greater detail.

FINANCE: The goal of having one year's operating budget in reserve was maintained last year when you consider the certificates of deposit in the headquarter's office account. It is hoped that this reserve can be kept at this level, as it should be, in the interests of good business practice.

THOUGHTS FOR THE FUTURE:

1. If individual members of this Association or any of the District Medical Societies and Specialty Societies are interested in having legislation introduced at any session of the State Legislative Assembly, this proposed legislation should be ready in bill form at least two months before the Legislature convenes.

2. Additional members from outside of Bismarck ought to volunteer for "Doctor of the Day" at the First Aid Station during the Legislative Session.

3. In our public relations program, personal contacts with persons and groups outside of organized medicine must be made and maintained.

ACKNOWLEDGMENTS: Your Executive Secretary wishes to express his sincere appreciation to our President, Glenn W. Toomey, M.D., for his efforts in behalf of this Association. Dr. Toomey made many trips both in and out of the State on Association business.

My sincere thanks also go to those other members with whom this writer has had occasion to work this past year on committee assignments and the several other programs of the association.

Respectfully submitted,
(Signed) Lyle A. Limond,
Executive Secretary

HOUSE OF DELEGATES INTERIM MEETING

December 11, 1966 Fargo, North Dakota

The Speaker of the House of Delegates, Dr. P. O. Dahl, called the meeting to order at 9:30 a.m. Roll was called by Dr. Buckingham, the Secretary, and the following delegates responded:

Doctors: B. F. Amidon, Fargo; M. H. Poindexter, Fargo; H. A. Norum, Fargo; A. C. Burt, Fargo; Paul Beithon, Wahpeton; R. J. Ulmer, Fargo; S. J. Cook, Alternate, Rolette; R. D. McBane, Devils Lake; J. W. Lind, Alternate, Bottineau; J. A. Sandmeyer, Grand Forks; G. L. Countryman, Grafton; K. S. Helenbolt, Grand Forks; H. W. Evans, Grand Forks; W. T. Powers, Grand Forks; V. J. Fischer, Alternate, Minot; Brian Briggs, Minot; A. M. Cameron, Minot; C. J. Klein, Alternate, Valley City; C. H. Peters, Alternate, Bismarck; M. A. K. Lommen, Bismarck; P. O. Dahl, Bismarck; E. O. Hieb, Jamestown; J. V. Miles, Jamestown; Dean Strinden, Alternate, Williston; W. C. Hanewald, Dickinson; R. E. Hankins, Mott; J. M. Little, Mayville.

There being a quorum present, Dr. Dahl declared the House of Delegates to be in session.

Speaker Dahl: This being an Interim Meeting of the House and there being no committees to bring the business before the House, the House of Delegates must act as if in a Committee of the whole.

Dr. Burt: I move that the House of Delegates act as if in a committee of the whole, during this session.

Dr. Sandmeyer: I second the motion.

Speaker Dahl: All in favor of this motion? All ayes. Motion passed.

We are meeting here today to study the pros and cons of legislation, and we are, therefore, meeting as a Committee to hear this material and take a stand on the motions introduced.

Article V of the Constitution states "The following shall have the privileges of the floor but without the right to vote; the President of the Association, the President-Elect, the Councillors, the Secretary, the Treasurer, the Past Presidents, and the Delegates to the American Medical Association."

I am sure there are persons who are knowledgeable in these areas, attending this meeting, who do not fit these categories, but whom we can give the privilege of the floor so we can hear them. The Chair will entertain a motion that we suspend the By-Laws so that we may give these people the privilege of speaking at this meeting.

Dr. Evans: I move that we suspend the rules of the By-Laws to extend the privileges of the floor to other doctors attending this meeting.

Dr. Burt: I second the motion.

Speaker Dahl: All in favor of this motion? All ayes. The rules are suspended as far as the privileges of the floor are concerned.

This Body, at the May Meeting, passed a resolution directing the Council to appoint a Committee or Foundation to put forth the Feasibility Study for a grant for Heart Disease, Cancer and Stroke, and when this Feasibility Study grant was obtained, to report back to this body before an Operational Grant was applied for. This has not been accomplished, but Dr. Christoferson will address this Body at this time, and if the House wishes to take any action, they may do so.

Dr. Christoferson: As your Speaker has pointed out, the House directed the Council at its May meeting to create a Foundation for the purpose of applying for a Feasibility Study Grant for the Heart Disease, Cancer and Stroke Program—under Law 89-239. Mr. Buchanan was hired by the Association this summer and part of his duties were to implement this non-profit foundation and get started on the regional advisory group in prep-

aration for this application. As a consequence of his activities, corporation articles were filed forming the North Dakota Medical Foundation. In addition to this, a regional advisory group was formed in eight categories. In the meantime, the officers of this organization were informed that a chance for success of a Feasibility Study Grant for North Dakota was quite impossible, the reason being the way the law was written; that there must be a medical school with a post-graduate medical educational program. This excluded North Dakota from any chance of obtaining a Feasibility Study Grant. Little more was done about this until North Dakota was invited by two regions to join their programs: Nebraska and South Dakota, and the Northland Regional Medical Program which encompasses at present just Minnesota. Both of these regions have completed their Feasibility Study Grant applications and have submitted them for approval. Both of them have invited North Dakota to become a part of their program.

It seemed necessary to have more information on this, so Dr. Toomey appointed an "Ad Hoc" Committee and appointed me as Chairman. I should further point out that in the interim period, the Nebraska group had invited Dean Harwood to be a member of their Board and he had also been invited to be a member of the Board of the Western States. He had also been invited to be a member of the Advisory Group of the Northlands. I also was invited to become a member of the Advisory Board of Northlands. I accepted this on a temporary basis, pending the future of North Dakota.

In any event, this Committee met just before the Council met, Friday night of this week. We had about a six-hour session on this matter of the application and our position on the general over-all position of the regional centers. I would like to orient you about this Committee.

No one really liked this legislation. They felt it was a certain infringement on medical practice. It was also the concensus that as long as it was passed, we should try to make as much of it as we could. The Medicare Act is an act that provides that things should be done in a certain way and dictates the certain way it should be done. The Act provided for these regional programs in a very liberal act. The AMA really wrote the Act in which they allowed tremendous latitude for each region. For this reason, it gave the regions a great deal of latitude in developing a program in improving health facilities and in the research. This, therefore, could work very much to the advantage of all of us, if it does do nothing else but pull us a little closer together in pooling our resources and talents in the better care of patients. The Committee feels we should proceed on our own under this non-profit organization that has been established, and draw up a Feasibility Study Grant application. To do this, we should have an inventory of all the available medical resources in our area; and should develop the lines of referral and the facilities that we have for the care of these heart disease, cancer and stroke patients; and as a result of this, the inventory should draw a conclusion as to the feasibility of developing these to a greater degree in the future. This is limitless, really, depending pretty much on what the individual wishes to do in the development of their program.

It was recommended by this Committee that the Program Coordinator be nominated for this and the Fiscal Director be nominated for this and that some financing be given so this could be done. This was brought before the Council on Saturday and they have approved the appointment of the Coordinator and the Medical School of North Dakota as a Fiscal Agent, and approved the idea of us going ahead and developing this application.

There was a dissent that perhaps we should not do anything about this, but the majority felt we should proceed on this from the standpoint of the Committee. The dissent was largely the feeling that by doing this we are playing into the hands of the Government and making ourselves more susceptible to regimentation of practice. Dr. Whisnant from Minnesota attended this meeting and the meeting of the Council and presented the Northland Regional Medical Program. At this time he pointed out that even though they would welcome North Dakota into the program, they would depend upon North Dakota to carry out a feasibility study. If North Dakota did not do this, and someone else did it for us, North Dakota would suffer.

What we would get out of this would be probably in direct proportion to what we would put into it from the standpoint of pointing up our own region as having certain advantages and facilities and drawing centers and medical centers, and pointing up the deficiencies and the needs we have for better medical care.

We have these proposals that the House should be aware of, if they care to take any action. The first proposal: that we proceed as we have been asking to do so, in the development of this Feasibility Study; Secondly—that we prepare to do this now. Or, should we join the regional program of Minnesota which is tailored to fit North Dakota; Or, should we join Nebraska which is able to accept North Dakota but has not included North Dakota in its original application; Or, should we drop the whole thing?

I think the action of the House was clear in its first decision. It is generally felt that North Dakota will never really be able to get an Operational Grant under this program of Heart Disease, Cancer and Stroke. I think it is generally felt that this legislation has far-reaching implications into the future of medical practice. We have the opportunity now of directing this in the proper manner, if we take the initiative to do so. If we drop the ball now, and become regimented in the future, it is our own fault.

It is anticipated that we will have to join some other region and I think the opinion is that we belong more to Northland Region; that we would have a better opportunity of getting our own oar in the stream and functioning with good representation on the Northland Regional Program, if we just made a Feasibility Study. If our application is not accepted, it is then anticipated that we could submit a supplemental application for a grant under Northland and they would get the money for North Dakota.

Dr. Dahl: Are there any questions of Dr. Christoferson? Thank you, Dr. Christoferson. The Committee will go ahead as directed by the House last spring and as determined by the Council Saturday. Does the House wish to initiate some action? If not, we will go on to the next order of business. This is Title No. 19 of the Medicare Bill, and the Carrier to be determined. Is there someone who would care to make a motion as to how to handle this regarding Title No. 19?

Dr. Hankins: I move that this Committee consider Title No. 19 legislation.

Dr. Powers: I second the motion.

Speaker Dahl: All in favor? All ayes. Motion passed. The Committee has before it now, Title No. 19, and the problem of a Carrier for this.

Dr. Burt: I would like to introduce this possibility. We are all very familiar with Title No. 18 and this presents no problem, which refers to all the people over 65. Apparently Congress felt that further Medicare should be offered to some of the rest of the people and

Title No. 19 was adopted rather hurriedly. This is an extension of the old Kerr-Mills Bill, which the Welfare Department was reluctant to permit to work. The states jumped on this, and it was the function of the states to determine the eligibility. We are familiar with what happened in New York state. Close to 50% of the population could be covered by this, and the estimated cost would be $275 million. This was the way the states were able to get money back from the Government. California started to do a similar thing, and this disturbed the Bureaucrats no end. No estimate was made that was reliable. The last figure that remains in my head is 18, but they are all going to get under Title No. 19. It is just a matter of time in our own state. This information has been run through computers and between Title No. 18 and No. 19, this will involve a third of the population of the state of North Dakota; Blue Cross takes care of another third, and the balance one-third is covered by other insurance carriers.

The danger of Title No. 19 is that this is the way of extending the control of medical practice. The fact is, this is built into the present law and requires no further implementation. Particularly in an election year, Congress produces a little bone for the taxpayer; so under Title No. 19 there is no reason why the age limit could not be reduced and other groups be taken in. Title No. 19 will socialize the medical profession over a period of time.

I would refer you to the report of the Medical Economics Committee who were unusually active this past year. The Welfare Department was there in force. It is difficult to get anything out of them on Title No. 19 in this connection. The carrier designation was extremely liberal. Anyone could apply as a Carrier if they had anything to do with the medical business; specifically, there was provision made for application of any State Welfare Department to be a carrier of either No. 18 or No. 19. Blue Shield is the Carrier for Title No. 18. The Welfare Department recognized the fact that this Title No. 19 was a big method for expansion, and I am sure whoever gets to be the carrier, Title No. 18 will fall to them. It was the feeling of the Medical Economics Committee and the feeling of the Medical Association as a whole, that they would, under no circumstances, desire to do business with the Welfare Department, if it can possibly be avoided. We have all been frustrated by dealing with the Welfare Department on an economic basis, either state-wide or locally. If there was any possible way to have some control of the designation of a Carrier for Title No. 19; if the Medical Association could influence the legislature to be specific about this, it would be very advantageous to the association as a whole. I do not know whether we should specifically designate a Carrier. I think we should open this up for discussion, as to how to go about this matter of eliminating our connection with the Welfare Department, 100%.

Speaker Dahl: The floor is still open for discussion. The Chair assumes that the question actually is that we consider legislation, if legislation is necessary, to have Blue Shield as the Carrier for Title No. 19; although it does not have to be. Would anyone care to move that this Body work with the Director of Blue Shield and form whatever legislation is necessary?

Dr. Richardson: I move that this Body recommend to the Legislative Committee that they, in conjunction with Blue Shield Board of Directors, formulate whatever legislation is necessary to support Blue Shield or some other private carrier for Title No. 19, other than the Welfare Department.

Dr. Powers: I second the motion.

Speaker Dahl: Is there any further discussion?

Dr. Burt: I tried to find out if there was someone in this House who knows more about this than I do. I am very much confused as to the legal implications of this. I understand, indirectly, that it is a function of the legislature, but does anyone know of anything as far as the law is concerned if this would be a function of something other than the legislature?

Dr. Peters: When we passed the Kerr-Mills legislation, in that Act there is a permissible carrier other than the Welfare Board. Assuming that this statute covers Title No. 19, this is already in the law as permissible, but not mandatory. I assume Dr. Burt is talking about direct billing. In Title No. 19, we are not able to bill directly as the statutes are written now. Those individuals under Title No. 19, who are also under Title No. 18, may be billed direct, but under the "Aid to Programs", our own legal staff says we can not bill directly, but it does mean a carrier outside the Welfare Board.

Dr. Burt: Is it true that Title No. 19 is almost an exact extension of Kerr-Mills?

Dr. Peters: Yes.

Dr. Fischer: Some of the things that have come up in Title No. 19 have been discussed in many areas. By 1975, all states must have this program in effect, or they will receive no Federal Aid. The North Dakota State Welfare Department is hoping to administer Title No. 19 funds.

Dr. Strinden: This is already being operated by the State Public Welfare. What we are asking is to transfer the function of the carrier from the Welfare Department.

Dr. Peters: There will be an effort by the Public Welfare to enlarge this to include greater areas.

Dr. Fischer: Would you please explain "medically indigent"?

Dr. Peters: The single individual may now have an income up to $1800.00 a year, or they can get up to $4000.00 a year as the number of the families go along, and still be considered "medically indigent", and would qualify under this program. This goes up to age 18. But the Welfare Board has plans to enlarge this if they can get appropriations so that it would include about 22% of the population of North Dakota. As an example, if someone had a bill of $900.00 for medical care, and their income was such that this would be over their allotment of finances subsistence, and supposing they could pay $150.00 of this, the Welfare Board would take care of $750.00. They would not compel the person to budget more than $50.00 per month to take care of that bill.

Speaker Dahl: May we hear from anyone who is opposed to the motion that is on the floor? If not, is the House ready for the question? All in favor? All ayes. Motion passed.

I have here a letter received from Dr. Lee A. Christoferson, dated December 8, 1966, as follows:

"As Chairman of the American College of Surgeons' Committee on Trauma for the State of North Dakota, I respectfully request that the House of Delegates of the North Dakota State Medical Association formulate a resolution recommending that the legislature of the State of North Dakota develop legislation requiring a licensing of all individuals operating a motorcycle or motor-bike, and providing certain safety standards in the equipment and operation of this type of vehicle. Recommendations of this legislation could be formulated by the Committee on Legislation of the North Dakota State Medical Association, and I will be glad to provide this. Committee with informational material from the National Safety Council and from other states where such legislation has already been passed.

Thank you very much for your attention to this matter.

Sincerely yours,
LEE A. CHRISTOFERSON, M.D."

May we have a motion that this communication be placed on the floor for discussion?

Dr. Ulmer: I so move.

Dr. Burt: I second the motion.

Speaker Dahl: All in favor? All ayes. Motion passed. We now have this letter from Dr. Christoferson up for discussion.

Dr. Christoferson: I think this is self-explanatory. The House should realize that the incidence of injuries among motor-bike operators is 4 to 1 in proportion to the number of vehicles operated. The number of these vehicles being sold is increasing, and they are being sold to young individuals who can become seriously injured. I mentioned this suggestion to one of our local dealers and he was vehement about his opposition to any legislation of this sort. I feel any of the physicians have a serious responsibility to this. I invited this man to come to the hospital to see these patients at any time. At one time we had five patients, all unconscious, who were injured seriously with these motor-bikes. I should mention that the National Safety Council considers this its number one item of business this year. The licensing of these drivers would simply mean it would cut out young, inexperienced drivers and the safety measure I would recommend is a helmet to protect the head from injury.

Dr. Ulmer: Are they not now required to have a driver's license?

Dr. Christoferson: I do not believe this is a law in the state of North Dakota.

Dr. Miles: Being the owner and operator of a motorcycle, I believe I can answer that question. In Jamestown I believe they have to be the proper age to get a vehicle license. However, the sad part of this is that you can not legislate against stupidity, or lack of intelligence in operating a vehicle. Perhaps the age needs to be increased as to when they can operate one. Even the car accident rates are higher with the teen-age group drivers.

Dr. Strinden: I know that youngsters have to have a license to operate a motorcycle, and the age limit is the same as for a car, at age 16. But, there should be some training programs to teach them to drive intelligently.

Speaker Dahl: Do you suppose it would be worthwhile that instead of introducing this ourselves, we write a Resolution to the State Highway Patrol, suggesting that they put through some legislation?

Dr. McLean: This was discussed this morning at the meeting of the Medical Advisory Council to the State Highway Department, the Safety Responsibility Division. Nothing had been planned. These people are required to be licensed with the same license as anyone driving an automobile. There are no further requirements. This has become one of the greater national responsibilities, as far as motorcycles are concerned. They are also required to wear headgear. We have had many of these Honda accidents in the smaller towns, also. I personally, feel we should have something from the medical profession. In one of our recent Medical News magazines there was an article on this, stating that the physicians have been very reluctant to get up and do anything about this. Only in the past year or two have the doctors been trying to get safety features for our cars. I do feel this is very important and that something should be done in North Dakota to protect these youngsters from these accidents.

Dr. Christoferson: I believe organized medicine should take the lead in protective medicine. One of the most serious tolls in human life are car accidents. This should be instituted on a legislative basis. Legislation can be passed which would improve these things. There are a lot of rental agencies who rent these to unlicensed drivers, and there should be a serious restriction against this kind of thing.

Dr. Miles: I was not speaking in opposition to this, but I feel it is as stupid to fly an airplane on a car license, as it is to drive a motorcycle on a car license.

Speaker Dahl: The discussion seems to be in favor of doing something about this. Are there any objections? Would anyone want to make a motion that we might act on this communication?

Dr. Beithon: I would move that the suggestions by Dr. Christoferson be referred to the Legislative Committee and that it be suggested that they prepare legislation which they can introduce at the next session, along these lines.

Dr. Strinden: I second the motion.

Speaker Dahl: This is now open for discussion.

Dr. Strinden: May I amend this motion to add that the Legislative Committee call on the Advisory Committee to the State Highway Department, Safety Responsibility Division, to help prepare this legislation?

Dr. Beithon: I will accept this amendment to the motion.

Dr. Amidon: I second this amendment to the motion, and would like to amend the motion to strike out the word "suggest" and insert the word "direct" the Legislative Committee to prepare this legislation.

Dr. Beithon: I will accept this amendment also to the original motion.

Dr. Strinden: I will also accept this amendment.

Speaker Dahl: We will now vote on the last amendment to the motion. All in favor? All ayes. Motion passed.

All in favor of the second amendment to the motion made by Dr. Strinden? All in favor? All ayes. Motion passed.

We will now vote on the original motion as amended. All in favor? All ayes. Motion passed. The original motion as amended is adopted.

We will now proceed to the next order of business which is the bill to be introduced for an act to amend Section 12-25-01; Procuring an Abortion—Punishment. I will accept a motion that this be placed on the floor for discussion.

Dr. Miles: I so move.

Dr. Hanewald: I second.

Speaker Dahl: All in favor? All ayes. Motion passed.

Dr. Buckingham: You have a copy of this bill before you and you will note that the only thing added is the phrase "or unless the woman became pregnant as a result of an incestuous act or rape", in Section 12-25-01; or as in Section 12-25-02: "or unless the mother became pregnant as a result of an incestuous act or rape". This is not a medical diagnosis, but is a legal diagnosis. This bill was balloted through the OB-Gyn. Society and it was 51 for, and 13 opposed.

Speaker Dahl: This is the present status in the North Dakota Code; the underlined material are the changes. Is there any discussion?

Dr. Amidon: I am on the Committee of Maternal and Child Welfare, and we really had some hassles about this. This is a small step towards legalizing abortion. We felt that if we tried to go too far, too fast, nothing would happen.

Dr. Ulmer: The First District Medical Society met and the feeling was expressed by a number of the members that we are not broadening it enough to cover some of the problems that exist.

Dr. Strinden: It is the feeling that we should go a little slow, or it will be a real problem for the physicians, because of people coming from out-of-state, should we

have in our laws those things not allowed in other states.

Speaker Dahl: Would anyone care to move what direction should be given to the legislative committee?

Dr. Lommen: I move that the House of Delegates advise the Legislative Committee to actively support this bill.

Dr. Little: I second this motion.

Dr. Ulmer: Who will introduce this bill?

Speaker Dahl: I think this one is from the Legislative Committee of the Medical Association. I know that our State Association drew up this bill, so it is up to the Legislative Committee to get someone to introduce it.

Dr. Ulmer: May I amend this motion to strike out the word "advise" and insert the word "direct" the Legislative Committee to support this bill.

Dr. Lommen: I will accept this amendment to my motion.

Dr. Little: I will also accept the amendment.

Speaker Dahl: All in favor of the motion as amended? All ayes. Motion passed.

You have before you a list of Public Health Legislation which is expected to be introduced in the next legislative session, and which the State Health Department would like to see passed. These are numbered from one to ten. These bills, however, are not written as yet. We will have this Body act on these as they see fit, and advise how they wish to handle this. May we have a motion to bring number one item to the floor for discussion?

Dr. Ulmer: I will move that we consider this.

Dr. Amidon: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed. This is now ready for discussion and comments.

Dr. Fischer: I went to a Governor's meeting on this regarding an appropriation for a children's and adolescent treatment unit. This meeting was well organized. There was no doubt as to what direction it was going to take. The Medical Association was invited to attend, but we had nothing to say about it.

Speaker Dahl: This is already in the mill. In 1961 our Association put out a pamphlet stating our Association's standing so that we are already on record as supporting this legislation.

Dr. Fox: I think this is more or less clear. In the past there has been some discussion as to whether this should go to Jamestown, or whether it should be put in a separate place, because the child or adolescent would have a bit of a taint being associated with Jamestown. The Senator from Grafton, who is more or less a majority leader in the Senate, is definitely opposed to having it in Jamestown.

Dr. Fischer: At the Governor's Conference, it was recommended that this be placed in Jamestown.

Speaker Dahl: We do not have any language of the bill, so it is very difficult to make a motion. However, may we have some motion on this?

Dr. Sandmeyer: I move that the House of Delegates go on record as approving the appropriation for a child and adolescent treatment unit in principle.

Dr. Hankins: I second this.

Dr. Miles: I appeared before the Senate appropriations committee regarding this treatment unit, and they thought it would be better to have the unit in Bismarck. The thought had nothing to do with geography or location, but was based on need.

Dr. Briggs: I am personally opposed to this as being a state unit for treatment of mental health diseases for children. We have in the community, mental health centers, and I cannot see why we want a state-operated center.

Dr. Beithon: I think we are already treating them at the State Hospital at Jamestown. They are trying to do this more effectively by segregating them in this intensive care unit and to get additional funds for staffing this.

Speaker Dahl: The motion originally was that we support this in principle, leaving it up to the Legislative Committee. Are you ready for the question? All in favor? All ayes, with the exception of one dissenting vote. Motion passed.

We will, with your permission, skip number two until later and take up number three, which is the Repeal of Section 12-33-02 which makes attempted suicide a crime.

Dr. Helenbolt: I move that this be discussed.

Dr. Amidon: I second the motion.

Dr. Dahl: All in favor? All ayes. Motion passed. Section three is now up for discussion. Would anyone care to explain this? I talked to our attorney last night and as this is the only reason he had to be here today, it was decided that it would not be necessary for him to appear at this meeting. In the present statute, attempted suicide as it stands today is a crime. In years past it was the thought that if it was made a crime it would stop suicides. Now the psychiatrists have decided that those who attempt to commit suicide should have treatment. This is to delete the words "makes attempted suicide a crime."

Dr. Ulmer: In Fargo there is no problem as we can refer them to our Neuropsychiatric Institute. However, in a small town this does pose a problem for where could the doctor put these people but in jail.

Dr. Strinden: Rather than to completely repeal this, I would suggest that they be held for psychiatric evaluation. There are instances where it is necessary to hold these people to insure that they be treated.

Speaker Dahl: I think we have to leave this up to the Legislative Committee.

Dr. Evans: What is the present punishment?

Speaker Dahl: Apparently the same as for attempted murder.

Dr. Evans: The psychiatrist has been notified that he is to report any attempted suicide, so every attempted suicide is reported, but there is no commitment or requirement for psychiatric treatment. I think it should be qualified.

Dr. Peters: One reason this is being proposed is that in Burleigh County there have been individuals who have attempted suicide and they have been prosecuted within the last few years. This is the reason for an attempt to repeal this law.

I move that this group should go on record as encouraging the Legislative Committee to support the repeal of this in principle.

Dr. Amidon: I second this.

Dr. Briggs: If we repeal this for people who are committing attempted suicide, how about the acts of these committed against others?

Speaker Dahl: That does not apply.

Dr. Little: I would like to see that the words "a crime" be omitted, but I would also like to have some way to hold these people because in most small towns we do not have a place to lock them up.

Speaker Dahl: I think this will come up under the number two item. All in favor of the motion as stated by Dr. Peters? All ayes. Motion passed.

We will, with your permission, skip number four, and take up number five regarding air pollution.

Dr. Beithon: I move that item number five be brought up for discussion.

Dr. Evans: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed. We do not have the bill, nor do we have information regarding this. Does anyone have a motion in regard to this?

Dr. Hankins: I move that we approve the authorization to establish a preventative air pollution program, and leave the final decision to the Legislative Committee.

Dr. Richardson: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed.

Dr. Powers: We are, it seems to me, wasting a lot of time here, trying to pass on a lot of things we do not fully understand because we do not have the bills. I believe the Legislative Committee are the ones that are going to have to look at the law and decide what is best.

Speaker Dahl: I agree with you, Dr. Powers. However, we are meeting today to take some action on these items. May we take up number six, which covers legislation to amend Section 25-09-07 relating to the procedure for collection of expenses for care and treatment of patients at the State Hospital.

Dr. Hanewald: I move that this be placed on the floor for discussion.

Dr. Fischer: I second the motion.

Speaker Dahl: All in favor? All ayes. Motion passed, and we will now discuss this item number six. I talked to Lyle Limond and the only information he has is that Dr. Amos apparently wants to have some type of bill so that he can keep track of expenses.

Dr. Peters: I move that this be tabled as not being within our sphere of interest or influcnce.

Dr. Miles: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed.

We will take up section number seven, regarding sanitation of restaurants, hotels and other public places.

Dr. Miles: I move that we place this on the floor for discussion.

Dr. Helenbolt: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed. As you know, we have two areas of inspection in this state for restaurants, hotels and other public places; such as the state laboratory and the State Health Department. I assume this is to take it from the state laboratory and place it in the State Health Department jurisdiction.

Dr. Ulmer: I move that we support the idea of having better sanitation, in principle.

Dr. Burt: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed.

We will take up number eight at this time. What are your wishes?

Dr. Helenbolt: I move that this be placed on the floor for discussion.

Dr. Miles: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed. It is now before the House of Delegates for the authorization and appropriation for a laboratory building. Is there anyone here who can give us some information on this?

Dr. Richardson: I do not know the details but I believe the Public Health Department is in favor of transferring all laboratory facilities and putting it in a separate building.

Dr. Powers: They feel very strongly on this. They are in favor of Grand Forks at the Medical School. At the University, the people feel very strongly that the State Laboratory should be in conjunction or connection with the Grand Forks Laboratory at the Medical School. I think we should certainly be in favor of a laboratory building.

Dr. Fischer: I do not feel that number eight is under our jurisdiction.

Dr. Powers: They are going to have to be moved out, and Dean Harwood in talking with the State Health Department mentioned that the Medical School was getting so crowded that they were going to have to look for quarters for the laboratory. If we are going to approve of anything, I think the House of Delegates should go on record as approving that the laboratory be in Grand Forks at the Medical School. I make a motion to this effect.

Dr. Evans: I second this.

Dr. Mahowald: I feel this is a part of medical education. Without this laboratory in Grand Forks, there would be a considerable amount of curtailment in the training of medical students and paramedical students. It is specifically a part of medical education. Fundamentally, the funds that are going into the Medical School are being used for this purpose. In addition to that, it is going to be extremely difficult to educate these people if there is no laboratory there.

Speaker Dahl: The motion before the House is that this Body direct the Legislative Committee to support legislation in this regard to keep the laboratory in Grand Forks. Is there any further discussion? All in favor? The ayes are in the majority. Motion passed.

There should be some clarification of this for the Legislative Committee. Are they not to support this, if it is not going to Grand Forks?

Dr. Powers: I think we have to do everything we can to support it, to go to Grand Forks, in principle, but if it can not be in Grand Forks we can not say it could not be any place. The Legislative Committee should do everything they can, but if it is not in Grand Forks, I do not want to say. We have to go by the wishes of the House. The Medical Association is in favor of this going to Grand Forks.

Speaker Dahl: Is there any disagreement with Dr. Power's statement?

We will now take up section number nine, the building of a 50 to ? bed facility to be located in Minot as a center for the study of the mentally retarded.

Dr. Burt: I move that this be discussed on this floor.

Dr. Amidon: I second this.

Dr. Ulmer: I move that we consider this as a unit and go over these items and support them or fight against them. I would ask permission to present this legislation that is being proposed relative to the mentally retarded.

Speaker Dahl: If there is no objection from the floor, the Chair will rule that Dr. Ulmer may go ahead.

Dr. Ulmer: (Communication from the North Dakota Association for Retarded Children, Inc.)

Your State Association Committee on Governmental Affairs met 11/12/66 at the NDARC office in Fargo for the purpose of discussing proposed legislation as it pertains to the mentally retarded of North Dakota.

In order to properly inform our local units, I will summarize the legislation proposed for the mentally retarded during the coming legislative session. I would appreciate any and all comments that your legislative committee chairman or member unit wishes to make on the legislation before this legislature.

I. It was the wishes of the Board of Administration and the LRC Committee to revise the present fee schedule for the Grafton State School. There are two new proposals presently being considered as possible legislation to go before the next legislative body. They are:

1. The proposal prepared by the Board of Administration.

A. A full fee will be paid by all parents with a forgiveness clause to be reviewed by the County Welfare Board. This program would be in effect till the child has been in the institution for 15 years or reaches his 21st birthday.

B. The ALC Church proposal and the NDARC proposal calls for the same charge allowed by the Internal Revenue standard deduction for a child with a forgiveness clause. This would be approximately 40% of the Board of Administration proposal. This would also be maintained till the child reaches age 21 or institutionalized for a period of 15 years.

II. STERILIZATION for institutionalized children only.

1. The institution determines that sterilization would benefit the patient.
2. The parent or legal guardian gives permission after being advised on the procedure of sterilization.
3. A five man committee reviews the findings. This committee would be composed of 3 physicians, a psychologist, a social worker.
4. The parent may also institute sterilization for their child in the institution using the same procedure that the institution must follow. Your legislative committee approved this voluntary sterilization act.

III. DEFICIENCY Appropriation. The LRC Bill would provide $50,000.00 to San Haven on an emergency act to provide finance for minor remodeling and furnishings and for additional staff to expand the San Haven facility to care for additional mentally retarded patients. This legislation does not call for major remodeling at the San Haven institution and should be considered a temporary measure. This bill was supported by the state committee.

IV. APPROPRIATION $100,000.00—to secure land and to study the future development of residential units in North Dakota. This institution will be in western North Dakota. There is no mention in the legislation as to the size of the institution. However, there are indications from some quarters that this institution should start at a size of 100 and expand to a size of 500. As you know, the national trend at the present time is to develop small community residential programs rather than large centralized institutions. Because there is no mention as to the size of the institution, the legislative committee approved action on this bill.

V. MANDATORY PKU BILL. Many states have a mandatory PKU testing bill. The Legislative Sub-Committee on social welfare determined that North Dakota should have such a bill to provide testing across the State of North Dakota. This legislation will not only provide for the testing, but also for public education and for further programs with metabolic causes so that as research continued, North Dakota can provide evaluations for children without new legislation. This bill was approved by your legislative committee.

VI. SUPERINTENDENT AND TEACHER RETIREMENT. At the present time the Superintendent at the Grafton State School is covered under teacher retirement. There is very little reason for this and most superintendents would not want to be covered by teacher retirement. The Sub-Committee drafted a bill removing the Superintendent of the Grafton State School from teacher retirement. Action approved by the legislative committee.

VII. GUARDIANSHIP AND GENERAL REVISION OF PRESENT CODE DEALING WITH THE MENTALLY RETARDED. The main discussion point on this rather large bill reviewing all of the code that deals with the mentally retarded in North Dakota gathered around guardianship at the institution. This bill is now drafted so that a parent may request the guardianship of the child until he reaches his 21st birthday and receives this legal status of the child. At age 21 the State School will notify the parent that he is no longer the guardian of the child but that he may request through court action to be given the guardianship of this child after age 21. If the parent does not take court action to obtain this guardianship, the Superintendent of the Grafton State School will be named the guardian of the child. General approval by the Committee.

VIII. DAY CARE PROPOSAL. This bill will not be introduced by LRC but will have individual sponsorship. Briefly, it will set state standards for Day Care for the mentally retarded and provide 50% matching funds to a local community carrying out a Day Care Program for the mentally retarded. Action approved by the Committee.

IX. SHELTERED WORKSHOPS. This legislation will be introduced by an individual legislator calling for 50% matching by the State of North Dakota to local community groups, providing a Sheltered Workshop. It will further set standards for the Sheltered Workshops in North Dakota. Action approved by the Committee.

X. RESIDENTIAL CARE. A bill might be proposed in the legislature by an individual legislator calling for state funds to equal that being paid at the largest institution in North Dakota to qualify community organizations providing a residential facility on the local community level. Action approved by the committee.

Dr. Ulmer: These last three items, namely, Day Care Proposal, Sheltered Workshops and Residential Care, would make it easier for organizations within communities to set up care, workshop shelters, and so forth. The laws of North Dakota say that the school board could set up adequate programs for retarded children. These have provided something, in some instances, for the mentally retarded but those up to six years old are not provided for. There is a school set up, called the "Opportunity School" and this is for Day Care. On these last three proposals the state would provide 50% of the funds to a local community carrying out these programs.

I move that the Legislative Committee study the bills that will be introduced, and that we support this in principle.

Dr. Countryman: I second this.

Speaker Dahl: There are 17 members of our Legislative Committee and there are five of them in this room at this time.

Dr. Powers: How will this compare with the operation at Grafton? Is there going to be a duplication on this?

Dr. Ulmer: At this time the Grafton School is full. This facility will be in Minot and not be in competition with Grafton. Many people in Grafton do not belong there in the first place. Now that they have been there, there is no place for them to go. The School at Grafton

will rule as to whether they will keep the child or not. The trend through the country is to reduce the size of these institutions and keep them at home to be in Sheltered Workshops or Day Care Units.

Dr. Countryman: This is fundamentally for the care of mentally retarded and not a study. A committee has been appointed to look into other state plans.

Speaker Dahl: All in favor of the motion as stated by Dr. Ulmer? All ayes. Motion carried.

This takes care of everything on this sheet with the exception of Items 2 and 4. I would like to cover Item 2, which is to amend Section 25-03-08 so that a physician can send an emergency mental case to a mental hospital without obtaining the consent from the county judge, or member of the county mental health board when none of these persons is readily available.

Dr. Ulmer: I move that this motion be considered on this floor.

Dr. Little: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed. This is now before the floor for discussion and action.

Dr. Little: Do we need this? Cannot we do it now? I have sent five people directly to the hospital and they have a mental board at the hospital who acts on this.

Dr. Ulmer: If these people are a voluntary admission, it can be handled that way. It is only a technicality about the charges that this is being brought up.

Speaker Dahl: This bill has not been written. There is a question of constitutionality. This is going to be gone over carefully by attorneys before they act.

Dr. Miles: The County Judge in our county leaves a signed permit on his desk, if he is going to be gone, in case this is needed.

Dr. Strinden: I think we should not make any recommendation. This should be turned over to the Legislative Committee. I move that we leave this completely up to the discretion of the Legislative Committee without any recommendations.

Dr. Fischer: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed.

The Chair will now call a recess for lunch, and reconvene at 12:45 p.m. in this room.

Dr. Mahowald: There will be no meeting of the Council after the House of Delegates meeting, today.

RECESS: 11:45 A.M.

At 12:45 p.m., there being a quorum present, Speaker Dahl called the meeting to order.

Speaker Dahl: The next order of business on the agenda is Item number 4, Promulgation of standards for, and licensing of laboratories in the State.

Doctor Hankins: I move that we place this on the floor for discussion.

Dr. Helenbolt: I second the motion.

Speaker Dahl: All those in favor? All ayes. Motion passed, and Item 4 is up for discussion. Are there any comments?

Dr. Richardson: I do not know why a copy of this bill has not been available to you as one has already been written. The law was written by the Legislative Research Committee on Public Health and requests a law for licensing laboratories and the licensing of the director or supervisor. The State Pathologist Association feels that licensing of state laboratories in North Dakota is not necessary because the mechanics for insuring the quality of medical practice in all its aspects, including clinical pathology, already are adequately provided for under the Medical Practice Act and implemented by the Board of Medical Examiners and that the proposed laboratory licensing law is a needless duplication.

The State Pathology Society passed a Resolution at their meeting, Saturday, December 10th. I would hope that this would be the attitude of the North Dakota State Medical Association towards this bill, with the supplement, that if we do not get our way, that we have something that we can live with to offer and support, so that we do not end up with something that is totally unpalatable to us. The motion would include this recommendation, that if we have to, we can use this bill as a guide-line, and the Legislative Committee would be given the authority and latitude to do the best they can. I can give the statement that I made at the third appearance in Bismarck regarding this bill before the Legislative Committee if you so desire.

RESOLUTION

WHEREAS, mechanisms for insuring the quality of medical practice in all its aspects, including clinical pathology, already are adequately provided for under the Medical Practice Act, and implemented by the Board of Medical Examiners, and,

WHEREAS, the North Dakota Society of Pathologists reaffirms its support of existing programs under the Joint Commission on Accreditation of Hospitals, and the College of American Pathologists, to upgrade lahoratory standards; and that the proposed laboratory licensing law currently being considered by the Legislative Research Committee is a needless duplication of these efforts;

THEREFORE, BE IT RESOLVED, that the North Dakota Society of Pathologists is opposed, in principle, to a laboratory licensing act at this time, and will continue in its efforts to insure that all laboratories providing services to patients in this state, meet *at least* minimal standards.

Signed: Gerald J. Obert, M.D.
Secretary-Treasurer; North Dakota
Society of Pathologists
December 10, 1966

I move that the House of Delegates of the North Dakota State Medical Association adopt this Resolution; changing the wording to read "The North Dakota State Medical Association".

Dr. Burt: I second the motion.

Dr. Countryman: Would Dr. Buckingham tell here from the bill what a "director" has to be and what a "supervisor" has to be?

Speaker Dahl: The bill involves any laboratory taking care of patients with more than two physicians that they must be licensed and they must have a director and a supervisor who are also licensed.

Dr. Buckingham: (read excerpts from the Bill, as follows).

"The director of a medical laboratory may not merely be nominal, but must participate actively in its operation. The director means an individual who administers the technical and scientific operation of a medical laboratory.

The supervisor of a medical laboratory means an individual who, under the general direction of the laboratory director, supervises technical personnel and reportings of findings, performs tasks, and in the absence of the director is held responsible for the proper performanee of all laboratory procedures."

"Qualifications of the Director of a Medical Laboratory"—

1—Be a physician certified in anatomical and/or clinical pathology by the American Board of Pathology.

2—or be a physician licensed to practice medicine in all its branches in North Dakota, or,

3—Holds an earned degree of doctor of science or doctor of philosophy and is certified by the American Board of Microbiology or the American Board of Clinical Chemistry.

"Qualifications of the Supervisor of a Medical Laboratory"—

1—Be a physician or has earned a doctoral degree from an accredited institution with a chemical, physical, or biological science as his major subject and, subsequent to graduation, has had at least 2 years experience in one of the laboratory specialties, or

2—Hold a degree of master of arts, or master of science from an accredited institution with a major in one of the chemical, physical, or biological sciences, and subsequent to graduation, has had at least 4 years of pertinent laboratory experience, or

3—Hold a degree of bachelor of arts or bachelor of science from an accredited institution with a major in one of the chemical, physical, or biological sciences, and subsequent to graduation, has had at least 6 years of pertinent laboratory experience, or

4—Be registered as a clinical laboratory technologist and have had at least 6 years of pertinent laboratory experience.

Dr. Richardson: You should probably know that the Public Health Service wants this bill for some reason. Even without the bill they already have this authority. If we can not kill this bill we should be in a position to instruct the Legislative Committee to try to come up with something we can live with.

Dr. Hankins: Which laboratories are involved?

Dr. Richardson: Every laboratory is involved in the state.

Speaker Dahl: All in favor of the motion by Dr. Richardson? All ayes. Motion carried.

Dr. Richardson: I would move that if the Legislative Committee and the people that are representing us before this legislature on this bill find that the bill cannot be gracefully killed, that they come up with something acceptable, using as a model the bill as written.

Dr. Burt: I second this.

Speaker Dahl: Is there any discussion? All in favor? All ayes. Motion passed. We now have the report of the Medical Education Committee with a supplemental report from Dean Harwood in which an appropriation is requested by the Medical School.

Dr. Ulmer: I move that we discuss this on the floor.

Dr. Amidon: I second the motion.

Speaker Dahl: All those in favor? All ayes. Motion carried.

Dr. Ulmer: I have here a copy of the Medical Education Committee Report and Dean Harwood's report. There are three courses of action possible, listed in these reports: 1—Attempt to have the mill levy raised; 2—Attempt to have the mill levy abolished and get all the funds through the regular University Budget; or 3—to keep what they have and ask for supplementary funds as needed.

The Committee recommends that activities be directed towards maintaining the mill levy in its present form and ask for supplementary funds as needed from the legislature.

Dr. Richardson: I move that this Body direct the Legislative Committee to support the Medical Education Committee's stand in the operation of the Medical School.

Dr. Burt: I second this motion.

Speaker Dahl: All those in favor? All ayes. Motion carried. The next item on the Agenda is the Student Loan Fund and the "Forgiveness" Clause.

Dr. Burt: I move that we bring this up for discussion.

Dr. Amidon: I second the motion.

Speaker Dahl: All in favor? All ayes. Motion passed.

Dr. Ulmer: Regarding the loan fund and the changes made in the law by the legilslature two years ago—the Committee felt that the State Medical Association should seek to have this law amended again this year to eliminate the size of the city that one must practice in for "forgiveness" ,purposes, and also to include residency training programs in the eligibility requirements. As you know, students may borrow up to $5,000.00 if they come back to practice in a community with less than 5000 population, and they are allowed "forgiveness" up to $1,000.00 per year. The Committee feels that this new amendment is deemed advisable in order to help in the establishment of additional residency training programs and also to attract physicians to the state since recruiting physicians for cities of all sizes has become a problem.

This means, therefore, that we include residency training under this bill of forgiveness—and that we remove the 5000 population limit on the city. We have great difficulty in getting people to come to practice in most any city in North Dakota. Any physician who comes back to practice in the state as a general practitioner, or as a resident, should be forgiven at the rate of $1,000.00 per year.

I move that we direct the Legislative Committee to introduce a bill to provide for "Forgiveness" for practicing in North Dakota either as a practitioner or as a resident, in any size city in which he wishes to practice. This would be an amendment to the present statute.

Dr. Burt: I second the motion.

Dr. Buckingham: I have approached several legislators already on this amendment. They were not from a small community, but they did not like this at all—that the doctor could practice in any city in North Dakota. This, of course, was a very small segment.

Dr. Little: I asked the rural legislators and they were very much against changing the 5000 population item. They felt the big cities had a lot of things to offer which the smaller communities do not have. I feel that if they are against this, we should not ask them as they might turn us down and when we ask them for something else, we would really like to have, they would not go along with us.

Dr. Strinden: Would it help if you included residency and instructors at the University, and leave out the 5000 population?

Dr. Ulmer: Perhaps this would help. It might be wise not to tamper with the 5000 population item.

Dr. Strinden: I would amend the motion to include the residents and instructors at the University, and to strike out the reference to the "under 5000 population community".

Dr. Hankins: I second the amendment to the motion.

We will now vote on the original motion as amended. All those in favor? All ayes. The original motion as amended is passed.

We now come to the communication from the Pharmaceutical Manufacturers Association regarding the "compulsory generic" drug measures which will be introduced in the 90th Congress. The legislation which some Senators support would restrict the use of trademark or brand-name prescription drugs and it would provide that all medicines for patients covered by such programs of the Social Security Act, must be "prescribed and furnished on a non-proprietary or generic basis".

Dr. Miles: I move that we discuss this on the floor.

Dr. Evans: I second this.

Speaker Dahl: All those in favor? All ayes. Motion passed.

Dr. Peters: I would like to speak on this. This topic has come up three or four times in the past three or four years. We have met with the manufacturers on this, also. It has been the position of the AMA Committee that we can not be opposed to prescribe drugs as cheap as possible; that we would be in an untenable position to oppose the generic prescription, but if we are compelled to prescribe these generic drugs, that these drugs are certified that they are effective drugs. The only control we have is a good pharmaceutical house for producing them. Actually we are basing our faith on this when we prescribe drugs under a trade-name. However, these should be certified that they are effective. But, from the long range on pharmaceutical research, if we are required to prescribe generic name drugs—where will the funds come from for pharmaceutical research.

Dr. Pederson: The House reaffirmed the present resolution that the physicians should be free to use the brand-name or the generic name. This resolution states in part "that we reaffirm the present policy of the Association, that physicians should be free to use either the generic or brand-name and encourage physicians to use medical judgment as to the effectiveness."

Dr. Strinden: I move that we use this Resolution as stated by Dr. Pederson.

Dr. Miles: I second this.

Dr. Burt: I think we should insist that this be spelled out. I would like to amend the motion regarding this resolution to the effect that the FDA certify the effectiveness and quality of drugs prescribed on a generic basis.

Dr. Helenbolt: I second the amendment.

Speaker Dahl: All in favor of the amendment to the original motion? All ayes. The amendment to the motion is passed.

All in favor of the original motion as amended? All ayes. The original motion as amended is passed.

Should a copy of this position go to our two congressmen and two senators?

Dr. Peters: Yes, and it should also go to C. Joseph Stetler.

Speaker Dahl: Dr. Buckingham has a few items to bring before the House at this time.

Dr. Buckingham: Is it your wish to have the Aid Station this year at the Legislature? We would appreciate it very much if you would send your name in and the date you can make it, to the Executive Secretary's office.

I passed out reprints from the J.A.M.A. regarding the Educational Background of Chiropractic School Faculties. You will note in this article that many of these fellows have degrees listed, but when the school is called, they have never heard of him. We would like to have you take these with you today and get them to the legislator in your area whom you know. Explain to the legislators that we do not want the chiropractors to have the name of "physician", and that is the gist of this.

Dr. Peters procured some forms and we will send them out to you so that you can get a profile on every legislator in your area—such as the name, age, family and pertinent facts regarding him. We will then have a file on every legislator. Please get these back to us as soon as possible.

Dr. Beithon: I would move that we continue to operate an Aid Station and reiterate that the doctors come out and help staff this.

Dr. Amidon: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed.

Dr. Buckingham: I also need permission from you men for you to go ahead and pass these pamphlets to the legislators. This is truthful information on the Chiropractic Schools.

Dr. Helenbolt: I move that permission be granted to pass out these pamphlets on Chiropractic Schools.

Dr. Hankins: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed. Dr. Buckingham is the Vice-Chairman of the Legislative Committee.

The next item on the Agenda is for Medicare payments on the part of deceased patients. If there has been a legal procedure to follow, Mr. Vogel advised that the next legislature should adopt a statute, making this less cumbersome.

Dr. Ulmer: I move that we bring this to the floor for discussion.

Dr. Amidon: I second this.

Dr. Burt: This is merely a technical question to ease the burden of payment under Medicare for the deceased patient. We are dealing with a very old-age group. This is going to be a cumbersome problem. If anything can be done to smooth the way, we think the medical profession should do so. I move that the Legislative Committee be sympathetic, if and when this legislation comes up.

Dr. Miles: I second this.

Speaker Dahl: All in favor? All ayes. Motion passed.

Dr. Ulmer received a communication from the North Dakota Academy of Ophthalmology and Otolaryngology on a resolution which they adopted. May we bring this to the floor for discussion?

Dr. Ulmer: I move that this be put on the floor for discussion.

Dr. Amidon: I second the motion.

Speaker Dahl: All in favor? All ayes. Motion passed. Dr. Ulmer, will you bring this to the attention of the House.

Dr. Ulmer: The following is the copy of the resolution:

The North Dakota Academy of Ophthalmology and Otolaryngology, a non-profit association of physicians and members of the NDSMA who specialize in the diagnosis, treatment—medical or surgical, and general care of the eyes, ears, nose and throat, and whose members are recognized as specialists in this field by the NDSMA, being fully cognizant of its responsibility to the care of patients in all fields of medical care encompassed by its members, does hereby resolve:

1. That all allied ancillary workers, such as orthoptic technicians, audiologists, dispensing opticians, etc., must be governed solely by the Medical Practice Act, and such laws as these ancillary workers are governed by must be incorporated only in that Act and no other Act.
2. That all ancillary workers, in the fields of Ophthalmology and Otolaryngology shall not be incorporated or included or governed by the acts or laws enacted by other professional or mechanically skilled practitioners.
3. That the North Dakota State Optometric Act remain as written, and that under no circumstances should that Act be broadened, to permit the use by optometrists of any drug instilled in or appliance applied to the eyes, or permit optometrists to diagnose, treat or recommend treatment of any eye condition other than those encompassed in the present North Dakota State Optometric Act.

This was sent to me by Dr. Williams who assured me that he had the approval of the Society for bringing it to your attention. The word was circulated around that

the Optometrists plan to put through a bill permitting them to put drops in the eyes and to diagnose and treat or recommend treatment of any eye condition.

Dr. Countryman: The optometrists claim that the ophthalmologists are only interested in prescribing contact lenses, but the optometrist says he fits the eye properly.

Dr. Ulmer: I would move that we support the position of the North Dakota Society of Ophthalmologists and Otolaryngologists.

Dr. Little: I second this.

Dr. Buckingham: I am confused on this. How are the opticians qualified to insert these contact lenses? What training have they had?

Dr. Ulmer: They manufacture them and fit them only on the prescription of the physician. I do not know what training they have had. Any ophthalmologist who prescribes these glasses will have a conversation with the optician about fitting these. The patient usually asks for names where they can go and they are given a list of names of opticians. The ophthalmologists want to be able to continue to prescribe contact lenses, but the optometrists want to be the only ones to prescribe and fit contact lenses. If they had their way, the opthalmologist would have to send the patient to an optometrist.

Dr. Ulmer: I am sorry that I did not insist that Dr. Williams come here today. There has been a continued battle between the Ophthalmologists and Optometrists. The optometrists want only to do this work and they are trying to prevent the ophthalmologists from refracting contact lenses.

Speaker Dahl: All those in favor? All ayes. Motion passed.

On the Agenda of the Council was a Statement of Policy by the North Dakota State Medical Association regarding the payment of usual, customary and reasonable fees under the ODMC "Medicare" program. In this it was stated that the recently published 1966 Relative Value Guide of the North Dakota State Medical Association is to be used in negotiating a new contract with officials of the Office for Dependents' Medical Care (ODMC) since the present contract expires on January 31, 1967.

Dr. Mahowald: The Directive was approved. The Council has approved the Relative Value Guide.

Dr. Burt: I move that this be brought up on the floor for discussion.

Dr. Richardson: I second this.

Speaker Dahl: All in favor? All ayes. Motion carried.

Dr. Fischer: The usual and customary fee, following the 1966 Relative Value Guide has never been approved by the House of the State Medical Association.

Dr. Lommen: I move that the House of Delegates approve the Relative Value Guide of 1966, that has been approved within the past few months.

Dr. Amidon: I second this.

Dr. Burt: There has been some misunderstanding about the 1966 Relative Value Guide. It was sent out to all the members of the Association. It is a guide, and has nothing to do with dollars and cents. All it has to do with is the relative value of all procedures. This was adopted because the old 1958 schedule was incomplete. Over the years, many things have been added. They are not being paid on a basis of relative value guide and they will not be. It was intended to be a guide as to relative value procedures.

Dr. Fischer: When this came out, the physicians were confused and used this as a schedule for Blue Shield. At no time has this ever been approved to be used as such. It has been a confusing thing to Blue Shield, also. This Body should adopt the Relative Value Guide of 1966 as proposed by the Economics Committee.

Speaker Dahl: All in favor? All ayes. Motion passed.

Speaker Dahl: Is there any further business to come before the House at this time?

Dr. Peters: The Speaker asked me to draw up a Resolution regarding Mr. Limond.

RESOLUTION

BE IT RESOLVED that this House of Delegates of the North Dakota State Medical Association in session on December 11, 1966, extends to its Executive Secretary, Mr. Lyle Limond, its sympathy for his unfortunate accident and hospital confinement, and that we missed his advice and counsel at this meeting, and, with his unboundless sense of humor in mind,

BE IT FURTHER RESOLVED that he not become accident prone; that he not unduly disrupt the hospital service, or the tranquillity of his physicians, and thereby his recovery be prompt and complete.

NOW THEREFORE, this House wishes him a speedy recovery and best wishes for A Merry Christmas and a Happy New Year,

AND BE IT FURTHER RESOLVED that the State Medical Association appreciates his sincere efforts in carrying on the duties of his office under handicap, and that we hope his legislative activities will be aided and assisted by an appropriate conveyance at the next legislative session.

I move the adoption of this Resolution.

Dr. Miles: I second the motion.

Speaker Dahl: All those in favor? All ayes. The Resolution is adopted unanimously.

I would like to introduce Mr. Sherwood Williams, the A.M.A. Representative, and extend the privileges of the floor to him, at this time.

Mr. Williams: It is always a pleasure to be at your meetings, and I would urge that North Dakota keep up the good work.

Speaker Dahl: Is there any further business? If not, I would like at this point to extend the appreciation and thanks of the House and of myself to Mrs. Fremming, Mrs. Kohler, and Mrs. Byrne for the efforts they have put into this meeting in making it run as smoothly as it has. We have been under the distinct disadvantage of not having our Executive Secretary present and these ladies have done a remarkable job in place of him.

Dr. Sandmeyer: I move that we adjourn.

Dr. Little: I second the motion.

Speaker Dahl: The House of Delegates stands adjourned.

ADJOURNMENT: 2:00 P.M.

REPORT OF THE CHAIRMAN OF THE COUNCIL

The First Session of the Council was held Thursday, April 28, 1966 in Fargo. The meeting was called to order at 7:00 p.m. by the Chairman, Dr. R. E. Mahowald. The Secretary, Dr. Keith Foster, being absent, Dr. Dean Strinden acted as Secretary and called the roll. The following Councillors responded:

Doctors: R. E. Mahowald, R. W. McLean, F. D. Naegeli, W. R. Fox, F. A. DeCesare, G. A. Dodds, G. Christianson, Dean Strinden, J. M. Van der Linde and J. J. Moses, proxy for R. B. Tudor.

Others present were: Doctors J. D. Craven, R. D. Nierling, P. O. Dahl, V. J. Fischer, G. W. Toomey, E. H. Boerth, R. C. Painter, Wm. Buckingham, and Mr. Lyle Limond and Mr. J. Sherwood Williams, Field Service Division of the A.M.A.

Dr. McLean moved, Dr. Strinden seconded, and motion passed, that the minutes of the previous meetings be approved as printed in the Agenda.

Dr. R. D. Nierling, Treasurer of the North Dakota State Medical Association, gave his financial report of January 1 to April 27, 1966. He explained the items listed on his financial statement and advised that he did not feel it would be necessary to raise the dues of the Association this year. The following report on dues collected and paid-up members was given by Dr. Nierling.

January 1, 1966 to April 27, 1966

Dist.	Reg. Members	Ret'd.	Ltd.	Total Pd.	Comp.	Hon.
First	91	6	–	97	–	5
Second	36	–	–	36	–	2
Third	78	2	–	80	–	4
Fourth	54	–	–	54	–	4
Fifth	11	–	–	11	–	1
Sixth	80	1	–	81	2	5
Seventh	29 1 ½ Yr.	1	2	33	–	1
Eighth	21	–	–	21	–	
Ninth	25	–	–	25	–	4
Tenth	10	–	–	10	–	–
	435 @ $100 ½ @ 50 @ $10	10	2 @ $10		2	26
	$43,500.00					
	50.00					
	100.00					
	20.00					
	$43,670.00 Total Amount					

A discussion followed, regarding the seating of Delegates in the House. Dr. Christoferson, Speaker of the House, advised that according to the report of paid-up members by Districts, the First, Third and Sixth Districts had elected one more delegate than they could seat in the House. Dr. Mahowald asked the Councillors of these Districts to check with their Secretaries as to the number of paid-up members and elected Delegates, so that the Speaker may seat the Delegates according to the Constitution.

The auditing of the books was brought up by Dr. Nierling. Motion was made in 1965 that this audit be handled by one firm. This has proved to be rather difficult and a slow process because of the necessity of sending vouchers, bank statements and books to Bismarck to be audited by one firm with Mr. Limond's statements and books. Dr. Mahowald suggested this matter be deliberated at the next meeting of the Council with the idea of reaching some satisfactory solution.

Dr. Mahowald introduced Mr. G. W. Smith, Branch Manager of Parke-Davis and Company. Mr. Smith presented to the North Dakota State Medical Association a set of framed prints depicting the History of Medicine. Dr. Craven accepted these for the Association and expressed the Association's pleasure and gratitude for this very fine gift. Dr. Strinden suggested that the Executive Secretary be instructed to write an appropriate letter of thanks to the President of this Company.

Dr. Amos was then introduced by Dr. Mahowald. He gave a brief talk on legislative bills that may come up on medicine, and asked for the doctors' support on one, S. 3008, and also requested that they lend their assistance to the Utilization Review Committees of the small hospitals.

Some discussion followed on the utilization of the hospitals. Dr. Christoferson advised that this matter would be brought up in the House of Delegates at their final meeting.

Mr. Donald Eagles, Executive Vice-President of the Blue Shield gave a brief summary of the Medicare Program, and the progress so far. Blue Shield was appointed a carrier for this program in February of 1966, with a 90-day contract. He discussed the problems Blue Shield has had at meetings with the Social Security Department, but at this time and at least for one year, the Blue Shield has the privilege of determining a "reasonable fee" to be used. At the present time, payments to be made to the doctors are to be made on the usual, customary schedule of fees to be charged. In gearing up for Medicare, Blue Shield has promoted one man on their staff as Coordinator of the Program and believe that it will take about twenty more employees to handle the program, and in line with this, the Government insists that the Blue Shield hire some people of a different color as employees.

Another item Mr. Eagles brought before the Council was that of a Liaison Committee with the North Dakota State Medical Association as to how far they can go in regard to reasonable fees. He feels it is very important to have such a committee to whom problems can be referred to and resolved.

Mr. Eagles advised that the Welfare Department has the privilege of "buying in" to this program, and welfare departments can qualify as Carriers for their recipients. North Dakota Welfare Department has not as yet "bought in". Mr. Ovre stated that they would operate as they have in the past and will wait to see what happens. Mr. Eagles hopes and feels they will be able to negotiate a contract satisfactory to all. They have a contract for 90 days, from February 11 to May 11. They can spend $10,000.00 to gear up for this program, but there is a clause that they can get the actual amount spent to gear up for this program. They will be going to Baltimore, Maryland the first part of May for conferences on this program and will have some help from the National Blue Shield Association legal staff at these conferences.

In regard to the Blue Shield Program in North Dakota, they have been planning for several years a change in the benefits, payments to doctors and premium payments. These have been held up until they get some idea as to the cost.

All of the information and papers they have received so far on Medicare is headed up "for discussion only". Nothing has, as yet, been decided upon. However, Mr. Eagles advised that the North Dakota State Medical Association could state that the Blue Shield program would be the one they recommend to handle the Welfare people in North Dakota. A discussion followed regarding the reasonable fee schedule. The Social Security Administration has not made any rules regarding the billing for Medicare patients. The difference between direct billing and assignment was explained and discussed for clarification.

Dr. Mahowald brought up the matter of the Podiatrist problem in North Dakota. The Podiatrists have sued Blue Shield, claiming that they were operating unconstitutionally by now allowing the podiatrist payment for doing work which he claimed he could do as well as any doctor of medicine. This problem was discussed by the Councillors.

Dr. Mahowald then called on Dr. Christoferson for information on Public Law 89-239, Heart Disease, Cancer and Stroke. Dr. Christoferson stated that the House had received no request for action on this item. After discussing this matter, Dr. Strinden moved, Dr. Moses seconded, and motion passed that the Speaker of the House be instructed to bring the matter of Heart Disease,

Cancer and Stroke, Public Law 89-239, before the House of Delegates for discussion. Dr. Christoferson stated that he would charge a Reference Committee with the recommendation to carry out some action or recommendation regarding this matter.

Dr. Mahowald then asked for information and discussion of a policy statement regarding fees charged persons in government programs, including Vista, Job Corps, Headstart, etc.

In answer to this, Mr. Limond discussed the correspondence he had received; a letter from Dr. Huntley of Minot to the Indian Health Area Director at Aberdeen, S.D., advising that the Medical Association feels they should be paid their usual and customary fees and in view of this, he could not sign the contract which was tendered for the care of the Indians. A copy of a letter from the Office For Dependents' Medical Care to Dr. Lindelow, Chairman of the Medical Economics Committee, in which it was suggested that the maximum allowance for tonsillectomy, adenoidectomy, dilation and curettage be modified so that the physicians in North Dakota could charge and be paid under the usual and customary fee concept. Another letter has arrived saying that they had surveyed the fees paid North Dakota physicians and noted that they would seem to be a little low in D and C, and T and A, and offering a higher fee which would be the top. It is, therefore, the feeling that the Government is going to go along and offered, without too much urging, to raise the fees in these categories. It is the feeling of the Economics Committee that the physicians should have the usual and customary fees for Vista, Job Corps, and Headstart.

After considerable discussion, it was decided by the Council that this should be referred to the Medical Economics Committee for their action, and these letters should be forwarded to the Secretary of the Association to be read before the House of Delegates. Dr. McLean moved that this matter be brought up on the floor of the House, and the recommendation by the Medical Economics Committee be reiterated and acted upon. Dr. Naegeli seconded the motion. Motion passed.

Dr. Mahowald then advised that Wisconsin will have their 125th Anniversary, and it is thought it might be a good gesture for the North Dakota State Medical Association to send a congratulatory telegram to the Wisconsin Medical Association signed by the President of the North Dakota Association. Dr. Naegeli moved that this be done; Dr. Christianson seconded, and motion passed.

A letter was then read by the Chairman of the Council, from Mrs. S. J. Thakor, President of the North Dakota Health Careers Council, asking for financial support to aid the Council in the production of a booklet to be distributed to junior and senior high school students; said booklet emphasizing the facilities in North Dakota for health careers. Mr. Sherwood Williams of the A.M.A. advised the Council that the A.M.A. has completed a booklet on this item, called "Horizons Unlimited"—which is available at no cost. He would send a booklet to the Council and one to Mrs. Thakor. Dr. DeCesare moved that this matter be tabled at the present time. Dr. Naegeli seconded. Motion passed.

Discussion then followed regarding the North Dakota Council as a Grievance Committee for the North Dakota Medical Association. Dr. Mahowald received a suggestion from Dr. Tudor, which was very well presented, on the problem of presenting a grievance to the proper committee. It was decided that an attempt should be made to define "grievances", such as "financial" or "professional". It was suggested that this be tabled until further thought could be given it, and to bring it up at a later session of the Council. Dr. Mahowald feels it is time for the Council to define what they would accept in the line of grievances and how they should be handled.

The Chairman of the Council discussed the matter of the Podiatrists of North Dakota. They have asked to have a Liaison Committee between the Medical Association and the North Dakota Association of Podiatry in the State which consists of six members. This request can be ignored, or the Council can recommend that a liaison committee be appointed by the President. After discussion, it was moved by Dr. DeCesare to table this item until a committee from the North Dakota Association of Podiatry could be met with. Dr. Fox seconded the motion and motion passed.

Dr. Christoferson, Speaker of the House, asked Dr. Naegeli, Dr. McLean and Dr. Christianson if their District Societies had submitted a list of candidates for the office of Councillor. This has been done and the list will be given to the Secretary, Dr. Buckingham.

Dr. Painter, Alternate Delegate to the American Medical Association, appeared before the Council and gave a brief talk on the proposed raise in dues of the A.M.A. He gave statistics of the increased expenses of the A.M.A. which makes it necessary to raise the dues. He asked for the undivided support of the members of this proposed raise. It was decided that this should be a matter of discussion for the House of Delegates.

There being no further business, the Council was adjourned, at 10:30 p.m., to reconvene on Sunday, May 1st, after the House of Delegates meeting.

To be continued in the September issue

Book Reviews . . .

Soviet Socialized Medicine: An Introduction

by MARK G. FIELD, *231 pp., The Free Press, New York, 1967. $6.95*

Important aspects of Soviet medicine are discussed in the following chapters: "Soviet Society and Its Health Service," "A Brief History of Russian Medicine Prior to 1917," "Marxism and the Ideology of Medicine," "The Development of the Soviet Health Service from 1917 to the Present," "Organization of the Soviet Health Service," "Health Personnel," "Clinical Facilities and Services," "Preventive Health Services," "Medical Research," and "Financing of the Health Service." The book is based on an exhaustive study of Soviet sources and several visits to Russia. The development of Soviet medicine is viewed in historical perspective, starting with Czarist Russia, and on the background of the political philosophy "Dialectic Materialism." Soviet medicine is, according to their sources and assumptions, theoretically superior to capitalistic medicine because the "Bolshevik Revolution eliminated the basic antagonistic contradictions between the socio-economic structure and the health of the people and thus did away with the basic source of illness for the workers." The emphasis of medicine, in line with the political philosophy, is on prevention, rather than therapy.

It is surprising to learn that in some influential circles it is seriously assumed that clinical medicine will finally wither away in a Soviet society. The development of Soviet medicine, between the goals of preventive and clinical medicine, has not followed a straight line, as this reviewer may add from his own experience in Russia. The neglect of clinical medicine in the late twenties and early thirties had disastrous consequences and clinical medicine was, thereafter perhaps temporarily, emphasized to a greater extent. However, an elaborate system of preventive services has been set up including regular check-ups of rather large segments of the population in "dispensaries." According to the number, type, and content of Soviet medical journals, it appears, however, to the reviewer that there is an overwhelming interest of the average Soviet physician, as in the West, in clinical rather than preventive medicine.

In 1965 the estimated total population of the U.S.S.R. was 229,100,000 with 443,300 physicians, 20,200 stomatologists, and 37,300 dentists. In addition, there are 1,523,400 semiprofessional health personnel, including 364,100 "feldshers" and 684,100 nurses. The occupation of a "feldsher," with training somewhat similar to that of a registered nurse, is unique to Soviet medicine and has a tradition of several hundred years. In smaller communities, feldshers take over responsibilities which are commonly reserved to physicians. The percentage of women physicians has grown from 10% in 1913 to 75% in 1963, that is, Soviet medicine is mainly a female occupation except at the top positions. The salary of an average physician with a seniority of twenty-five years is 108 new rubles per month, technically equivalent to about $100, although not directly comparable. In any case, the salary is very modest and not attractive for more ambitious students. Top administrative and particularly academic positions are much better paid, however.

The crucial question is whether fifty years after the revolution Soviet socialized medicine provides better service than medicine in "capitalist countries." According to the information given in this book, such is not the case. As in industry, "norms" are given in terms of visits (consultations) per hour, for example, 5 patients per hour in an outpatient clinic in internal medicine, 8 in ophthalmology, and so forth. From the experience of the reviewer it appears likely that the physician who sees more patients than the "norm" requires will be rewarded, like the industrial worker who exceeds his norm (one can count patients, but cannot easily evaluate the thoroughness or effectiveness of attention). The average Soviet physician appears to have a shorter working day, much more paper work, and less technical help than his "capitalist" colleague. There is no indication that the absence of payment by the patient has improved the relationship between the physician and the patient but this is admittedly hard evidence to find. The author does not attempt a qualitative evaluation of Soviet medicine, but this would be crucial for a comparison. In addition to the number of beds, number of hospitals, and number of medical personnel, it would have been helpful to also give some data on treatment periods, disability and mortality rates, equipment and service in hospitals such as coronary care units, number and type of diagnostic tests per patient in various diseases, development of new therapeutic and diagnostic procedures, and development of new drugs.

The author states modestly that the main purpose of the book is introductory, but it is actually quite comprehensive and informative, with one exception of the section on medical research having a condensed treatment of 14 pages.

The book is highly recommended because medicine in Russia represents the extreme of socialized medicine which we must be informed about.

ERNST SIMONSON, M.D.
Minneapolis

Anticancer Agents

by FRANCES E. KNOCK, M.D., *272 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $15.50*

In an attempt to improve the methods of cancer chemotherapy to patients with advanced malignant disease, the authors have presented the related aspects of surgical-chemical treatment of cancer at preclinical and clinical levels. In a large part, the book is a plea for patient-centered cancer therapy and for coordinated surgical-chemical treatment of cancer, individualized with the chemical requirements of patient's cancer cells. No attempt was made to encompass all the forms of cancer therapy, though in some instances, particular emphasis has been made on some recent advances in each field. The author has drawn upon broad reference material, though this has not unduly weighted the text with bibliographical detail.

(Continued on page 14A)

(Continued from page 13A)

As a result of the method of presentation of the material, some aspects of the problem of cancer chemotherapy are unduly emphasized, while others lack the necessary detail desirable for the practicing physician or student. The heavy emphasis on the sulfhydryl inhibitors seems to reflect the author's personal interests in this field of cancer therapy. In the specific details of some drugs, the author has concisely summarized the mechanism of action and the role of the agent, but left it incomplete as to the method of administration and guidelines to the use of such drugs. If this had been done, many sections would have been more valuable to the reader.

In general, this publication is one of the new attempts to present material relating to cancer chemotherapy agents in such a manner that the practicing physician and student can have some reference point to obtain a summary of these agents. The students in learning have found it difficult to find an appropriate reference where all the chemical agents that are of practical use have been described. This book approaches this aim. Hopefully, this type of approach will provide a stimulus for more complete textbooks on cancer chemotherapies that can be used as a manual for the practical treatment of patients with advanced malignant disease.

B. J. KENNEDY, M.D.
Minneapolis

Neurological and Neurosurgical Nursing: An Introduction

by DONALD F. SCOTT, M.B., *and* BARBARA DODD, S.R.N., *204 pp., ill., Pergamon Press, New York, 1966. $6*

A concise paperbound book on the general concepts of neurological and neurosurgical nursing which would be helpful to the graduate nurse with limited experience in neurological nursing. The style is lucid, easy to read, and well organized. Written in an informative and concise manner, it should serve as a basic outline for the care of patients with neurological problems. The anatomical and physiological aspects and the clinical features of the more common neurological problems are briefly presented in conjunction with medical management, surgical intervention, and the proper identification of nursing objectives.

There are 15 well-planned chapters beginning with the "Admission." This deals with pertinent nursing observations to be made, a detailed review of the neurological examination, and the lumbar puncture. Chapter 2 deals with the care of the patient with a brain tumor with excellent correlation of the clinical features, anatomy, special diagnostic studies, and the pre- and postoperative nursing aspects required with surgical intervention. Again, this same logical approach is used in covering the diseases relating to the vascular system of the brain. Chapters 5 and 6 deal with the head injury patients and care of the unconscious patient. The remaining chapters 7 through 13 briefly cover the more common neurological and neurosurgical disorders ranging from epilepsy, degenerative and infectious diseases of the nervous system, diseases of the extrapyramidal system, muscular diseases, and myasthenia gravis. Chapter 14 briefly encompasses pathology involving the peripheral nervous system and surgical procedures for relief of pain in patients with malignant diseases. The "Discharge of the Patient" is the final chapter which too briefly includes the roles and contributions of the family and social worker in discharge planning.

According to the preface, the authors intended this book to be used as a guide for nursing students. It would be informative and useful to the graduate nurse practicing on a medical or rehabilitation floor as well as in a neurological or neurosurgical unit. The extensive glossary would be a valuable addition to the bookshelf of any nurse in neurology or neurosurgery. Numerous and excellent black and white illustrations add to the book's usefulness.

MARGARET CLIPPER, R.N.
Minneapolis

Reticuloendothelioses in Childhood: A Clinical Survey

by HAROLD W. BARGEON, M.D., *127 pp., ill., Charles C Thomas Co., Springfield, Ill., 1966. $8.50*

As a result of the association of the author with a long experience with the reticuloendothelioses, he has reviewed some of the known properties of the cell in the normal, and more particularly in the diseased state. The literature that has accumulated on the subject of the reticuloendothelial system is enormous.

In the introduction, the basic knowledge of the system is presented, and a discussion ensues of the problems that exist in the full understanding of these diseases. The major portion of the book discusses the specific disorders of the system, giving particular attention to the clinical aspects of the noninfectious, nonlipid group, to which the term "reticuloendotheliosis" has been applied. A clinical review of 64 cases is presented. There is particular emphasis on the less clearly identified reticuloendothelioses designated as eosinophilic granuloma of bone, Hand-Schuller-Christian disease, and Letterer-Siwe disease. A sound basis for diagnosis and prognosis is established. The therapeutic modality used in treating these diseases and dosages for the most common chemotherapeutic agents that are effective in the management of this group of diseases are given.

The author concludes with an optimistic future: that the investigator, student, and clinician will all contribute to the increased cures and diminished mortality rate of all the reticuloendothelioses.

B. J. KENNEDY, M.D.
Minneapolis

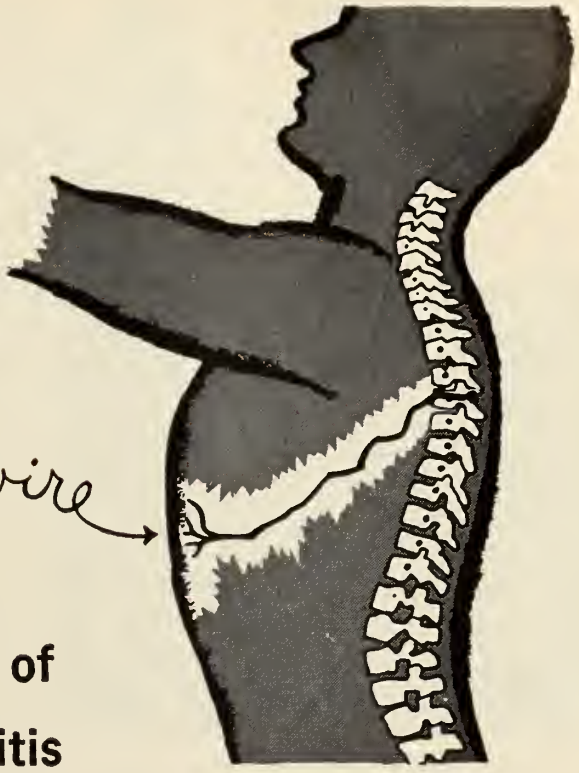
Radiology in World War II

edited by KENNETH D. A. ALLEN, M.D., *1132 pp., ill., U.S. Government Printing Office, Washington, D.C., 1966. $8.25*

No more dramatic example of the impact of modern medical science and technique can be found than the X-ray. Discovered three years before the outbreak of the Spanish-American War, the X-ray found its first use in United States military medicine during that conflict. But military surgeons considered its use limited until the United States involved itself in World War I. Radiology in 1918 was in an early stage of development; equipment, especially glass plates, was inadequate and in short demand and the number of radiologists was small. Fortunately, during the period between World War I and World War II radiology became a speciality,

(Continued on page 16A)

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—Boxes of 10 ampuls, 1.3 cc. each.—

References: (1) Baker, A. G.: Penn. Med. J. *63*:697-698 (May) 1960. (2) Smith, R. T.: New York Med. *8*:16-19 (Aug. 20) 1952. (3) Smith, R. T.: Med. Clin. N. America (March) 1957. (4) Lehrer, H. W.; Lehrer, H. G., and Lehrer, D. R.: Northw. Med. *54*:1249-1252 (Nov.) 1955. (5) Sforzolini, G. S.: Arch Ophthal. *62*-381-385 (Sept.) 1959. (6) Shupala, E.: W. Virginia Med. J. *56*:191-193 (June) 1960. (7) Xander, G. W.: Western Med. *1*:14-17 (Sept.) 1960. (8) Combs, F. C., and Canizares, O.: New York J. Med. (Mar. 15) 1952, pp. 706-708. (9) Marsh, W. C.: U.S. Armed Forces Med. J. *1*:1045 (Sept.) 1950. (10) Love, T. R.: Rocky Mountain Med. J. *50*:873 (Nov.) 1953.

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(Continued from page 14A)

equipment became perfected, and formal training produced radiologists with skill and talent. In short, the X-ray, at the outbreak of the second world war, was a well-established diagnostic and therapeutic tool.

This massive, detailed volume, a fitting companion to other volumes comprising the official history of the medical department of the United States Army during World War II, thoroughly delineates the role that radiology played. It is a vast mine of information not only for those interested professionally in radiology but also for historians concerned with the development of modern medicine. Each major and minor theater of operations, from the large Mediterranean to such small areas as Trinidad and the Persian Gulf, is described with reference to administration, personnel, facilities and equipment, diagnostic considerations, and noncombat diseases and conditions. Among the latter are tropical diseases, malnutrition, and backache. A variety of radiologists set down special experiences.

An entire section is devoted to radiology and the atomic bomb, including discussions of radiation therapy, tracer studies and growth of literature, dosage tolerance, and remarks on the development of health-safety programs. Curricula for the intensive basic course in roentgenology for medical officers at the Walter Reed General Hospital and for the basic course in X-ray techniques for enlisted men of the Army school of roentgenology at the University of Tennessee are printed in the appendixes. More than 300 well-selected illustrations accompany the text, and 8 charts, 14 maps, and 14 tables contribute to the volume's worth and usefulness.

PHILIP D. JORDAN, PH.D.
Minneapolis

International Encylopedia of Pharmacology and Therapeutics, Section 12: Ganglionic Blocking and Stimulating Agents: Vol. 1: Muscarinic and Nicotinic Stimulant Actions at Autonomic Ganglia

by R. L. VOLLE, *110 pp., ill., Pergamon Press, New York City, 1966. $5*

This series of books has been planned by a group of leading pharmacologists to meet the need for an expansive presentation covering the whole field of pharmacology. The overall plan calls for about 200 volumes organized into some 84 sections, with individual monographs in each section being available separately.

Dr. Volle covers the muscarinic and nicotinic stimulant actions at automatic ganglia in eight well-written, brief, but inclusive chapters. The first is a short historical survey, the second an excellent and lucid discussion of ganglionic cholinoceptive sites. The next two chapters cover the actions of nicotine-like and muscarine-like drugs, followed by chapters on a comparison of adrenal medulla with sympathetic ganglia, the action of 5-hydroxytryptamine, histamine, polypeptides, and catecholamines. The last chapter is a lucid survey of the physiological implications. The chemistry and physical properties of the drugs are given together with the metabolism and pharmacological and toxicologic aspects. Every chapter is followed by a comprehensive bibliography.

In these days of chemotherapy, the actions of drugs on autonomic ganglia are of great importance. This book is short and the style is so easy to read that even the busy practitioner can take the time to brush up on the subject. Yet it goes into the problems in sufficient depth

to be of value to the researcher. The book is highly recommended to both. If it is a fair sample of the rest of this encyclopedia, the editorial board is to be congratulated on a great service to pharmacology.

DAVID B. SABINE
New York City

The Cervical Syndrome

by RUTH JACKSON, M.D., *333 pp., ill., 3rd ed., Charles C Thomas, Springfield, Ill. 1966. $10.50*

In this era of rapidly increasing automobile accidents, with resultant neck injuries, the subject of this book becomes increasingly important to physicians. The author's vast experience in dealing with the cervical syndrome is readily apparent. No attempt will be made by this reviewer to point out all the good features of this book, of which there are many. The initial anatomy section is not only comprehensive but well illustrated. The sections dealing with etiology, symptomatology, and radiographic examination are also excellent. The author covers aspects of prevention as well as conservative and surgical treatment in the final chapter. One cannot overlook the excellent bibliography which is current and extensive. This book should be read by all orthopedic residents and physicians dealing with accident victims. It is a worthy addition to the bookshelf at a modest investment.

H. J. ERICKSON, M.D.
Minneapolis

NEW BOOKS RECEIVED

Foot Disorders: Medical and Surgical Management
by NICHOLAS J. GIANNESTRAS, M.D., 521 pp., ill., Lea & Febiger, Philadelphia, 1967. $22

Proceedings of the 2nd Meeting of the Federation of European Biochemical Societies, Vienna Volume 1: Antibodies to Biologically Active Molecules
edited by BERNHARD CINADER, 424 pp., ill., Pergamon Press, New York, 1967. $18.50

Inhaled Particles and Vapours II: Proceedings of an International Symposium, British Occupational Hygiene Society, Cambridge, 26 September - 1 October, 1965
edited by C. N. DAVIES, 605 pp., ill., Pergamon Press, 1967. $22.50

Inborn Disorders of Sphingolipid Metabolism: Proceedings of the Third International Symposium on the Cerebral Sphingolipidoses
edited by STANLEY M. ARONSON and BRUNO W. VOLK, 513 pp., ill., Pergamon Press, New York, 1967. $20

Androgens, Virilization and the Hirsute Female
by EUGENE J. SEGRE, M.D., 102 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $6.75

Techniques of Autoradiography
by ANDREW W. ROGERS, 335 pp., ill., Elsevier Publishing Co., Inc., New York, 1967. $20

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News Briefs . . .

North Dakota

DR. GALEN JAY EASH of Casselton was elected to active membership in the American Academy of General Practice.

* * * *

DR. W. E. G. LANCASTER of Fargo has resigned from the Cass County Mental Health Board. Dr. D. H. Lawrence of Fargo was named as his successor.

* * * *

DR. HERBERT J. WILSON of New Town exchanged practices from June 19 through July 14 with Dr. Edward W. Armstrong of Bethel, Vt., Dr. Wilson's ancestral home. Both doctors are the lone physicians in their communities.

* * * *

DR. PETER MORRISON returned to Mandan from Scotland and resumed his practice, also succeeding his partner, Dr. Shelagh Milligan, who will be leaving Mandan.

* * * *

DR. LEONARD W. LARSON of Bismarck was awarded an honorary LL.D. degree at the University of North Dakota, where he was the commencement speaker. He is a former president of the American Medical Association.

* * * *

ELECTED at the annual meeting of the North Dakota Heart Association were Dr. J. F. Houghton, Fargo, president; Dr. Kenneth W. Kihle, Bottineau, president-elect; Dr. T. H. Harwood, dean of the University of North Dakota Medical School, Grand Forks, vice-president; and Drs. Charles Hamilton, Bismarck, Harold Evans, Grand Forks, and C. J. Klein, Valley City, directors.

Minnesota

DR. J. J. LENARZ, who has practiced medicine in Brownerville and neighboring communities since 1936, was honored June 11 by a Dr. J. J. Lenarz Day in Brownerville. Dr. Lenarz is a native of Albany, Minn.

* * * *

DRS. CLARENCE JACOBSON, Chisholm, and DONALD MCCARTHY, Minneapolis, received Distinguished Service Awards from the Minnesota State Medical Association at the association's annual banquet in Duluth.

* * * *

DR. A. H. FIELD of Burnsville, a Farmington area family physician and surgeon since 1940, has accepted a position, beginning Sept. 1, with the Student Health Department of the University of Arizona.

* * * *

ELECTED at the 114th annual convention of the Minnesota State Medical Association were Dr. O. L. N. Nelson, Minneapolis, president; Dr. O. M. Heiberg, Worthington, first vice-president; Dr. H. J. Walder, Duluth, second vice-president; and Dr. Robert Flon, St. Paul, secretary. Reelected were Dr. Charles A. Neumeister, Minneapolis, treasurer, and Dr. J. T. Pewters, Minneapolis, speaker of the association's House of Delegates.

* * * *

DR. NORMAN HOOVER of the Mayo Clinic, Rochester, left for Saigon, South Vietnam, where he will be field direc-

tor of a joint U.S.-South Vietnamese program for improving medical education under the auspices of the U.S. Agency for International Development (AID). The program is administered by the American Medical Association, which named Dr. Hoover to the post.

* * * *

REELECTED at the annual meeting of Blue Shield of Minnesota was Dr. Charles A. Neumeister, Edina, president. Elected were Dr. Charles M. Bagley, Duluth, vice-president; Dr. Ellery M. James, Mahtomedi, secretary; Dr. John G. Lohmann, Pipestone, treasurer; and Drs. Bror F. Pearson, Shakopee, and A. Harold Mork, Anoka, directors.

* * * *

DR. JAMES H. MOLLER, assistant professor of pediatrics at the University of Minnesota, received a 1967 Minnesota Medical Foundation Distinguished Faculty Teaching Award, which includes a cash prize of $1,000.

* * * *

DR. C. WALTON LILLEHEI, University of Minnesota professor of surgery and world-famous heart surgeon, will assume the chairmanship of the surgery department of Cornell University Medical College in New York City, Nov. 1. He will also become the Lewis Atterbury Stimson professor of surgery at Cornell and surgeon-in-chief of the New York Hospital, the New York City general hospital that serves as a teaching institution for Cornell.

* * * *

DR. EDWARD BANNER of the Mayo Clinic, Rochester, was named president-elect of the Continental Gynecologic Society, which is composed of specialists in obstetrics and gynecology from the United States and four foreign countries.

DR. ROBERT KIERLAND, head of the section of dermatology at the Mayo Clinic, Rochester, was reelected secretary of the American Dermatological Association.

* * * *

DR. THOMAS G. MURN was elected chief of the St. Cloud Hospital medical staff succeeding Dr. Edward M. LaFond.

* * * *

DR. A. E. CARLSON, a Warren physician since 1933, closed his practice to become senior physician at the Veterans Administration Hospital at Fargo, N.D.

* * * *

DR. MARIO FISCHER, executive health officer of St. Louis County, retired and was succeeded by Dr. A. J. Houglum, deputy county health officer. Dr. Fischer, who became Duluth's first city public health officer in 1932, had held the county post since 1959 when the city and county health departments were combined.

* * * *

DR. C. J. STADEM, Twin Valley physician for the past sixteen years, terminated his practice to join the Crookston Clinic.

* * * *

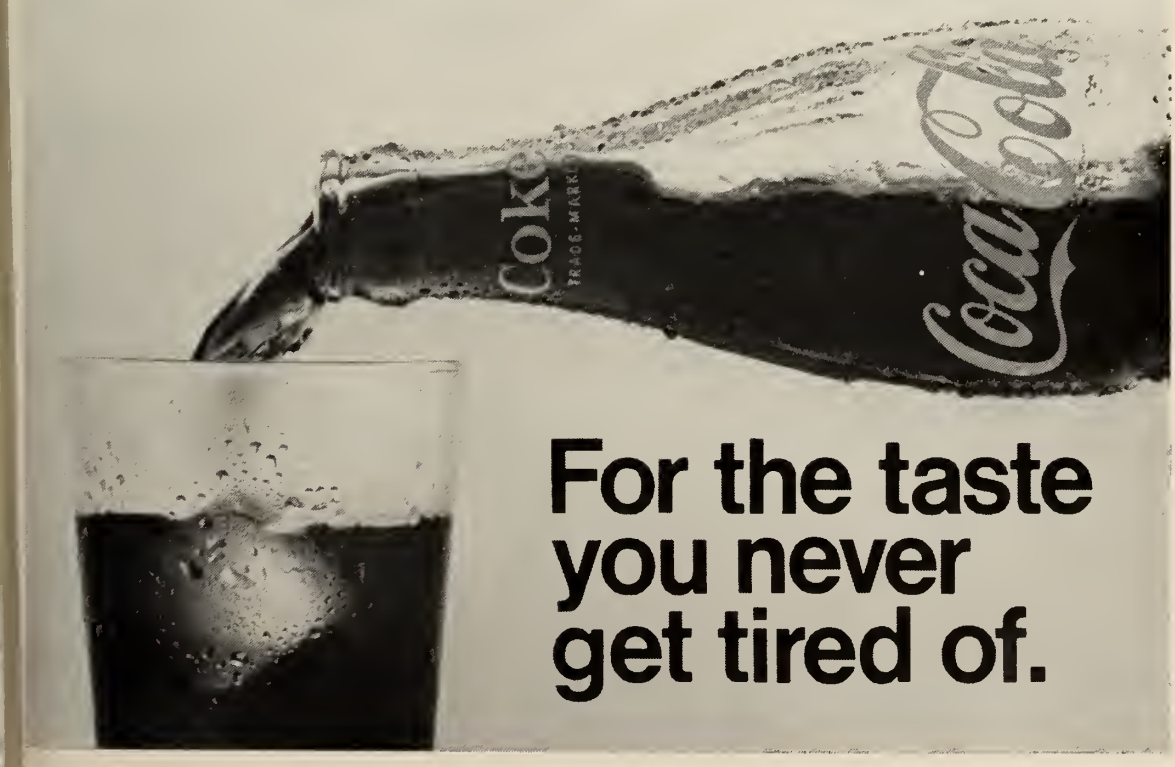
DR. KRISTOFER HAGEN of Edina, who has served twice as a medical missionary in India, was elected president of the Twin Cities' Chapter of the Lutheran Medical Mission Association. He is medical adviser to the Board of World Missions of the Lutheran Church in America.

* * * *

DR. FRED KANE, medical director and the first full-time psychiatrist at the Minnesota Residential Treatment Center for mentally disturbed children at Lino Lakes, resigned because of "frustration with complex administrative problems."

(Continued on page 22A)

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NEWS BRIEFS

(Continued from page 21A)

DR. JOHN WATKINS of Wells was elected to the board of directors of Planned Future Life Insurance Company.

* * * *

DR. BENJAMIN MC CALLISTER of the Mayo Clinic, Rochester, was elected to membership in the American Society of Internal Medicine.

* * * *

DR. WILLIAM REMINE of the Mayo Clinic, Rochester, was named president-elect of the Priestley Society. Composed of former first assistants of Dr. James Priestley, Mayo surgeon and former chairman of the Clinic's board of governors, the society is dedicated to furthering the knowledge of surgery.

* * * *

DR. RICHARD O. LUNDBORG, formerly of Madison, was honored for outstanding performance in the Mayo Graduate School of Medicine at Rochester. Dr. Lundborg, who received an Edward John Noble Foundation award for display and development of leadership, is now a consultant in anesthesiology at Mayo Clinic.

* * * *

DRS. ANDREW SINAMARK, Hibbing, and ALEXANDER E. VENABLES, Ely, were honored by the Minnesota State Medical Association for fifty years of service to the medical profession.

* * * *

DR. E. P. BRYANT resigned from the medical staff of the Faribault State School to resume his practice in the Ellendale Community Clinic on a full-time basis.

* * * *

DR. ROBERT PIERCE of Mountain Lake is also practicing medicine on a part-time basis in Heron Lake.

* * * *

DR. DONALD BERGLUND of Northfield was awarded a plaque by the Minnesota Association for Retarded Children in recognition of his many years of work for the retarded on local, state, and national levels.

South Dakota

DR. ROBERT H. HAYES of Winner was presented with a Superior Service Award at the National Institutes of Health, Bethesda, Md. The award was for outstanding service while Dr. Hayes was the medical officer in charge of the Public Health Service Surgical Mission in Nha Trang, South Vietnam, from January 1966 to January 1967.

* * * *

DR. EDUARDO G. FRANCISCO of Estelline was elected to active membership in the American Academy of General Practice.

* * * *

DR. JAMES DAGGETT was scheduled to begin practice in Lennox on July 1.

* * * *

ELECTED at the annual meeting of the South Dakota Heart Association were Dr. Gerald Tracy, Watertown, president-elect and director; Drs. William B. Janss, Rapid City, and Bruce Lushbough, Brookings, vice-presidents; and Drs. R. A. Buchanan, Huron, D. Max Reade, Yankton, H. Streeter Shining, Rapid City, Harold E. Lowe, Mobridge, and M. E. Sanders, Redfield, direc-

tors. Dr. T. J. Wrage, Watertown, past president, was given a recognition award for meritorious service.

* * * *

ELECTED by the South Dakota Medical Association were Dr. John Stransky, Watertown, president; Dr. John T. Elston, Rapid City, president-elect; Dr. Robert H. Quinn, Sioux Falls, vice-president; Dr. A. P. Reding, Marion, secretary-treasurer; and Dr. Robert Hayes, Vermillion, speaker of the House. Dr. Reding also was named delegate to the American Medical Association and Dr. Quinn, alternate.

* * * *

HONORED at the annual awards banquet of the South Dakota Medical Association were Dr. G. Robert Bartron, Watertown, who was presented with the Association's Community Service Award; Dr. Paul Hohm, Huron, who received a past president's plaque; and Dr. John Tschetter, Huron, who was recognized for his fifty-years of practice as a physician in South Dakota.

* * * *

DR. JOSEPH KOVARIK opened a practice in urology in Rapid City. Two physician brothers of Dr. Kovarik, Richard A. and Wenzel J., are also engaged in medical practice in Rapid City.

Deaths . . .

DR. ALFRED T. KAPSNER, 53, Princeton, Minn., died June 2. He had served three years in the Army Medical Corps during World War II and had practiced in Princeton since 1946.

* * * *

DR. GEORGE R. MC NEAR, JR., 44, Heron Lake, died May 27. He had been associated with the Mankato Clinic from 1953 to 1964, when he began general practice in Heron Lake.

* * * *

DR. EDWARD DYER ANDERSON, 75, a former president of the Hennepin County Medical Society, Minneapolis, died in Rougemont, Switzerland, May 27. He had served as assistant and associate professor of pediatrics at the University of Minnesota from 1918 to 1954.

* * * *

DR. ALVIN P. SCHEIB, 80, Watertown, S.D., died May 22. A pioneer South Dakota physician, he had practiced between 1908 and 1948 in Capa, Phoebe, Hitchcock, Elkton, and Watertown.

* * * *

DR. FREDERICK GRIEBENOW, 89, Bismarck, N.D., died June 5. He had been a member of the staff at Quain and Ramstad Clinic following his graduation from the University of Minnesota Medical School in 1909.

* * * *

DR. JOHN DWIGHT KEYES, 64, Winona, Minn., died May 23. He had practiced in Winona from 1930 to 1960 and was also a physician and surgeon for the Chicago and Northwestern Railway.

* * * *

DR. JOHN DE J. PEMBERTON, 80, Rochester, Minn., died May 18. He had been a pioneer in goiter surgery at Mayo Clinic, from which he retired in 1952 after a career of thirty four years as head of a section of surgery.

Classified Advertisements

RATES: 70 words or less, one time, $3. two consecutive times, $5. Copy must reach us by the 10th of the month preceding publication. REPLIES to advertisements with key numbers should be addressed in care of THE JOURNAL-LANCET, 65th at Valley View Road, Southdale Park, Minneapolis, Minnesota 55424.

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GENERAL PRACTITIONER WANTED—group of five desires another general practitioner. East Central North Dakota. Excellent school system and recreational facilities. Write. C. J. Klein, M.D., P.O. Box 911, Valley City, North Dakota.

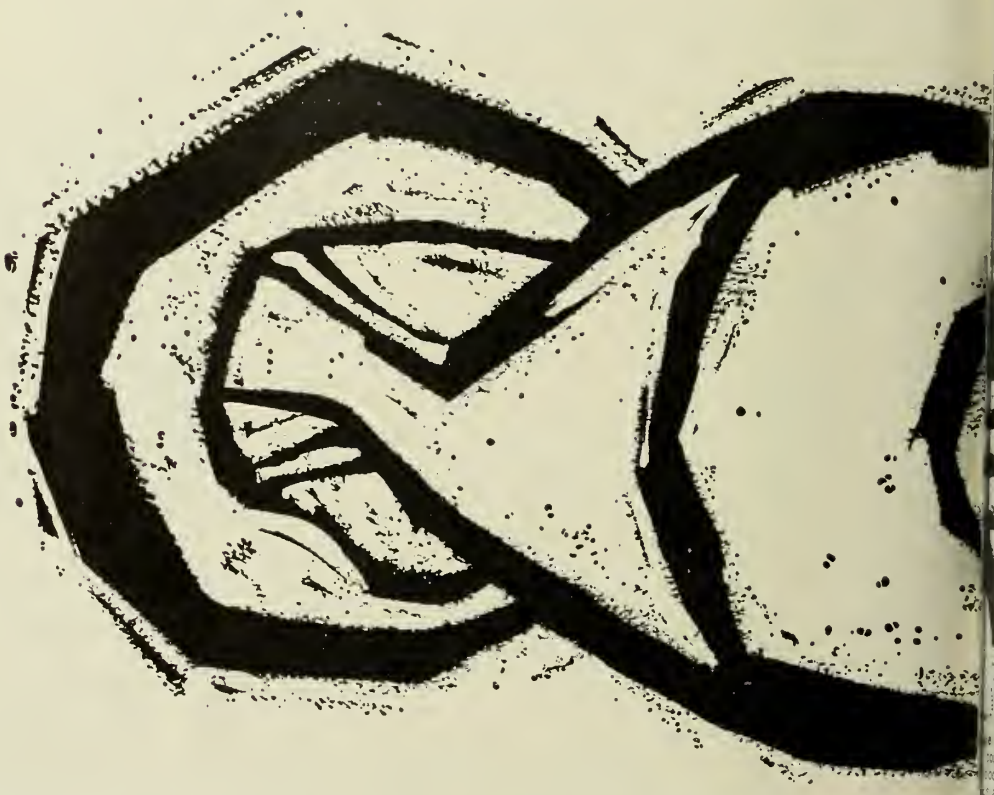
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Precautions: Administer with caution in severe ulcerative disease because of predisposition to perforation of bowel wall. In advanced chronic disease with loss of mucosa and fibrosis, steroids might hasten deterioration. In severe cases, it is hazardous to delay surgery more than a few days awaiting response to medical treatment. Because of possible adrenal insufficiency, therapy must be withdrawn gradually. If patient is subjected to unusual stress while on steroid therapy or within a year thereafter, adequate supportive measures and increased or reinstated steroids are indicated. For surgery, these measures must be continued throughout pre- and postoperative recovery periods, bearing in mind possible deleterious effect on fresh intestinal anastomoses. Steroids might impair surgical prognosis by increasing hazard of infection. If infection is suspected, administer appropriate antibiotic in large doses. Observe all precautions common to other steroid therapy. Watch for growth suppression in children during long-term use. Keep patients under close observation. Discontinue treatment and institute appropriate measures, if severe reactions occur. If necessary to use Cortenema in pregnancy, examine infants closely for hypoadrenalism; if signs are seen, institute appropriate measures, including steroids.

Adverse Reactions: May produce all side effects that occur with other forms of hydrocortisone, such as moon face, buffalo hump, fluid retention, excessive appetite and weight gain, abnormal fat deposits, mental symptoms, hypertrichosis, acne, striae, ecchymosis, increased sweating, pigmentation, dry scaly skin, thinning scalp hair, thrombophlebitis, decreased resistance to infection, negative nitrogen balance with delayed bone and wound healing, menstrual disorders, neuropathy, peptic ulcer, decreased glucose tolerance, hypopotassemia, adrenal insufficiency, necrotizing angiitis, hypertension, pancreatitis and increased intraocular pressure.

In children, suppression of growth may occur. Increased intracranial pressure may occur and possibly account for headache, insomnia, and fatigue. Subcapsular cataracts may result from prolonged usage. Long-term use may result in catabolic effects characterized by negative protein and calcium balance. Osteoporosis, spontaneous fractures and aseptic necrosis of hip and humerus may occur as part of catabolic phenomenon.

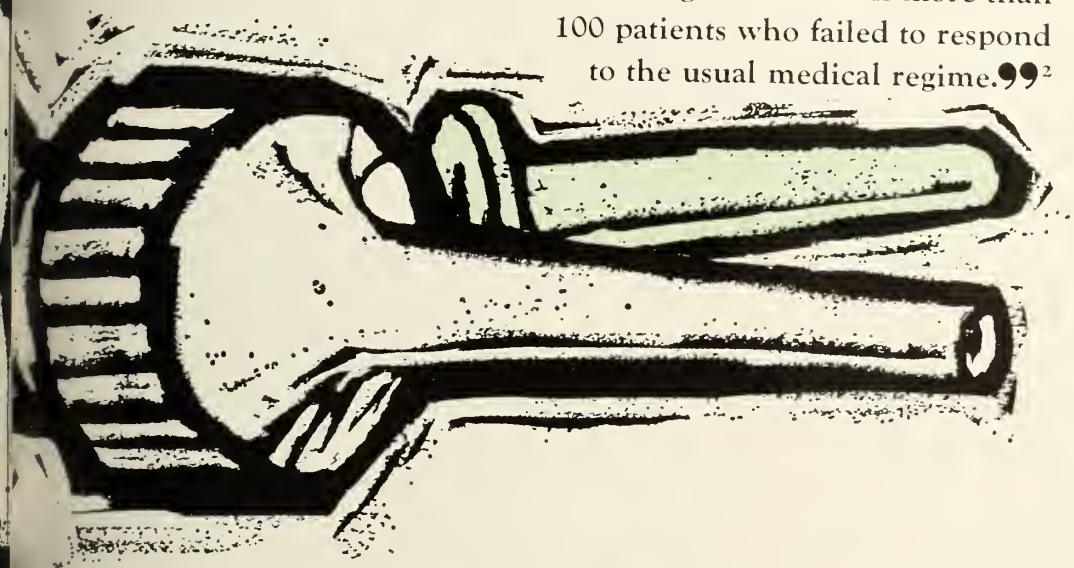
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Instruct patient to lie on left side during instillation. Shake bottle vigorously to resuspend hydrocortisone. Expose lubricated tip by removal of sheath. *Carefully* insert tip into rectum in direction of sacrum. Slowly express contents by compressing container. After instillation, patient should remain on left side at least 30 minutes.

Duration of treatment depends on degree of response. Symptomatic response usually occurs within 5 to 7 days. Improvement in mucosa, gauged by barium enemas and sigmoidoscopic examinations, may lag somewhat behind clinical improvement. Usual duration is two weeks, although some cases require up to 2 or 3 months. Minimal control of symptoms is insufficient basis for prolonged use. If no clinical or proctologic response occurs within 2 or 3 weeks, or if patient's condition worsens, discontinue use.

Symptomatic improvement should not be sole criterion in judging efficacy. Actual sigmoidoscopic examination and X-ray visualization are most reliable. Since steroids can inhibit wound healing, Cortenema should not be used in immediate or early postoperative period following ileorectostomy.

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1. Sherman, L. F.: The treatment of ulcerative colitis with hydrocortisone alcohol retention enemas. A topical and systemic anti-inflammatory agent, *Minn. Med.* 48:605, May 1965.

2. Sherman, L. F., Tenner, R. J. and Schottler, J. L.: Treatment of ulcerative colitis with hydrocortisone alcohol retention enemas: A topical and systemic anti-inflammatory agent. Part II, *Dis. Colon & Rectum* 10:43, January-February 1967.

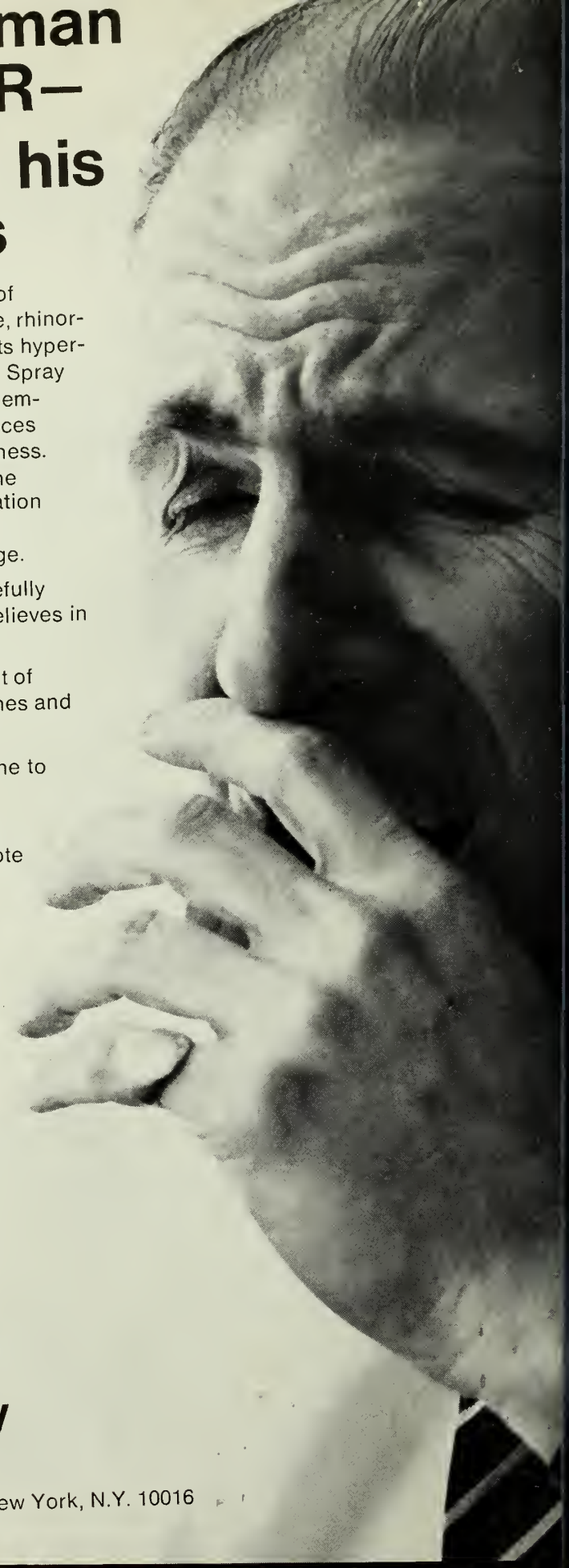
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CODEINE TO SUBDUE COUGH remains the best antitussive and appears to act synergistically with PAPAVERINE in the relief of cold symptoms—and to enhance aspirin analgesia.

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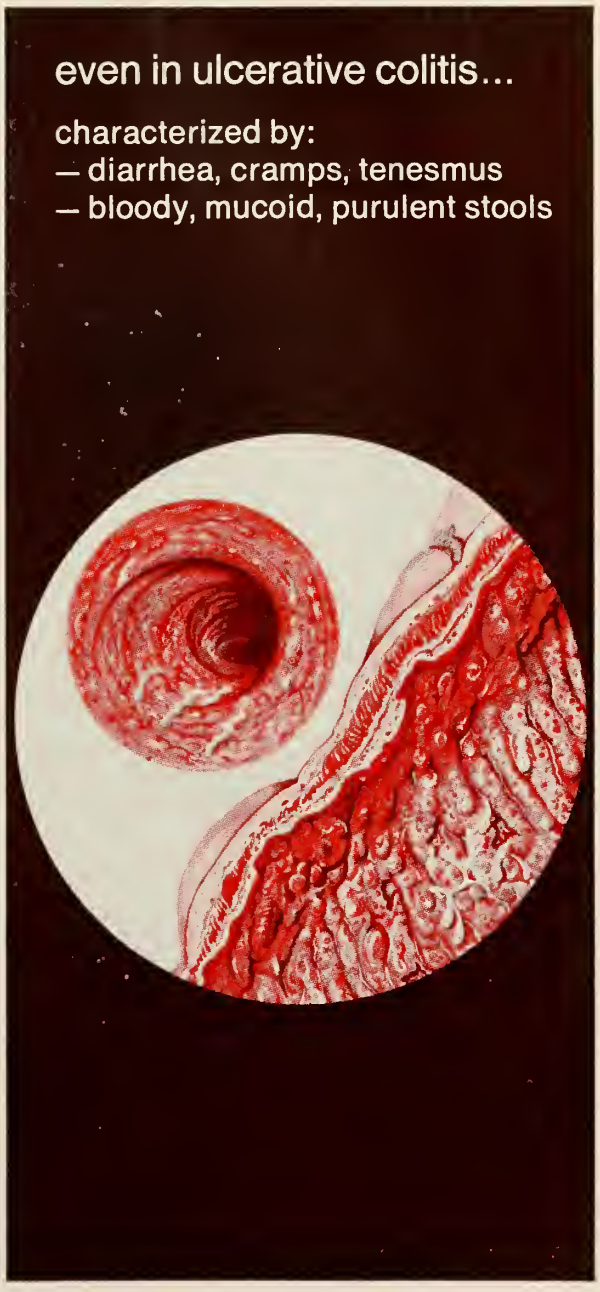
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3-6 mo. . ½ tsp.* t.i.d. (3 mg.)
6-12 mo. . ½ tsp. q.i.d. (4 mg.)
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8-12 yr. . . 1 tsp. 5 times daily (10 mg.)
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*Based on 4 cc. per teaspoonful.
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Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of the extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.



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The Journal-Lancet Volume 87, No. 9, SEPTEMBER 1967

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CONTENTS

Incidence of Rheumatic Fever: Summary of an Eight Year Study of Incoming Freshmen at the University of North Dakota . . . 317
Edwin G. Olmstead, M.D., and Jane Churchill

Results from Shunting Procedures for Internal Hydrocephalus . . . 321
Lee A. Christoferson, M.D.

Functional Problems Confronting the Obstetrician . . . 325
Irving C. Bernstein, M.D.

Surgery—Yesterday and Tomorrow . . . 328
Owen H. Wangensteen, M.D.

Transactions of the North Dakota State Medical Association . . . 333

Book Reviews . . . 12A

News Briefs . . . 17A

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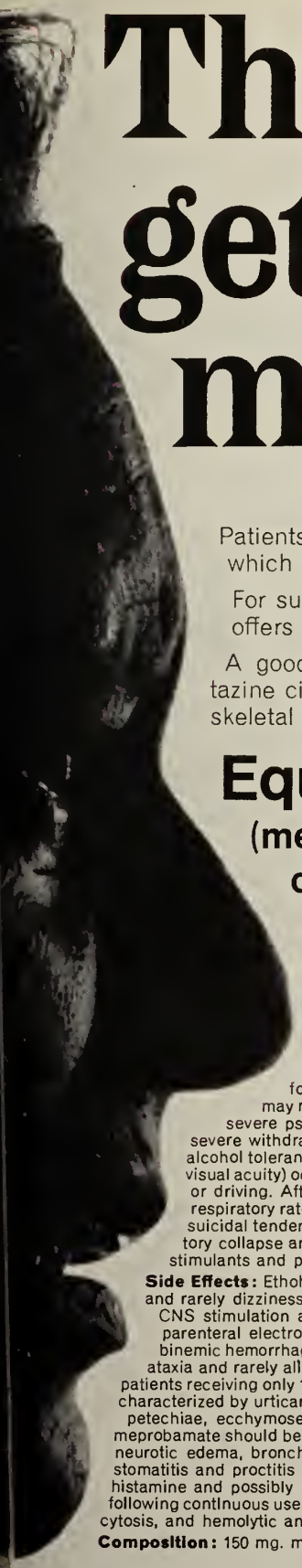
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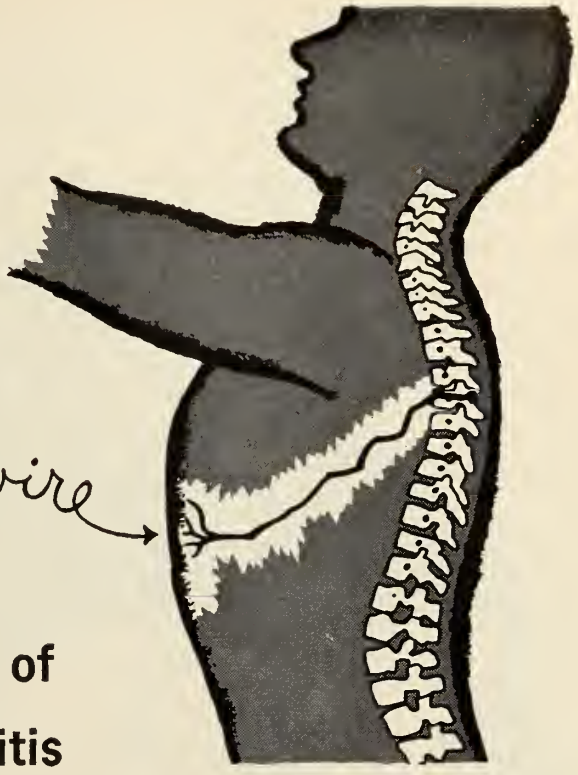
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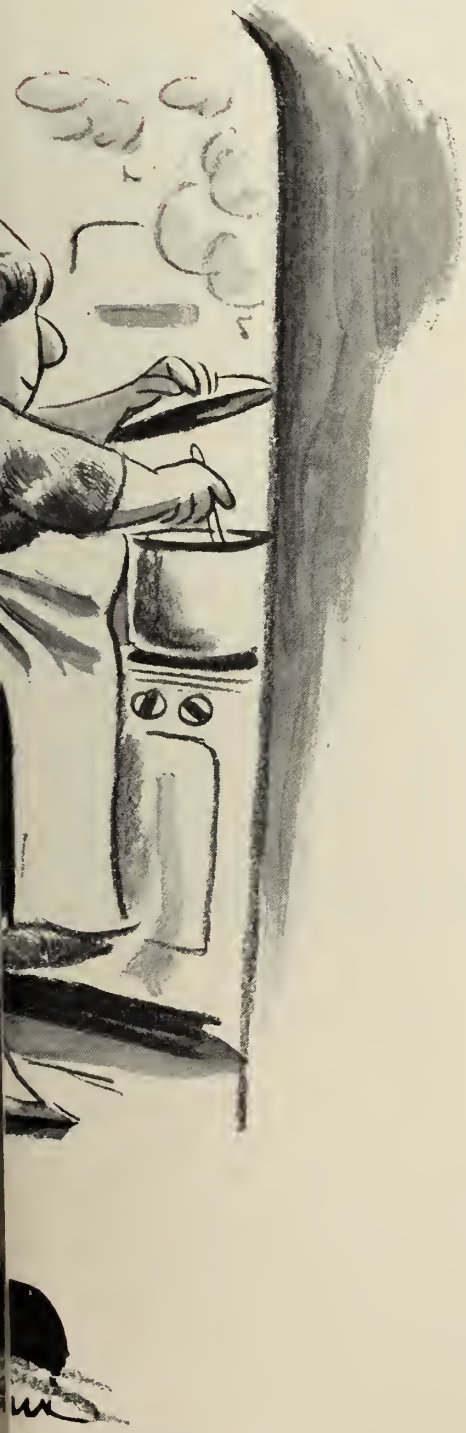
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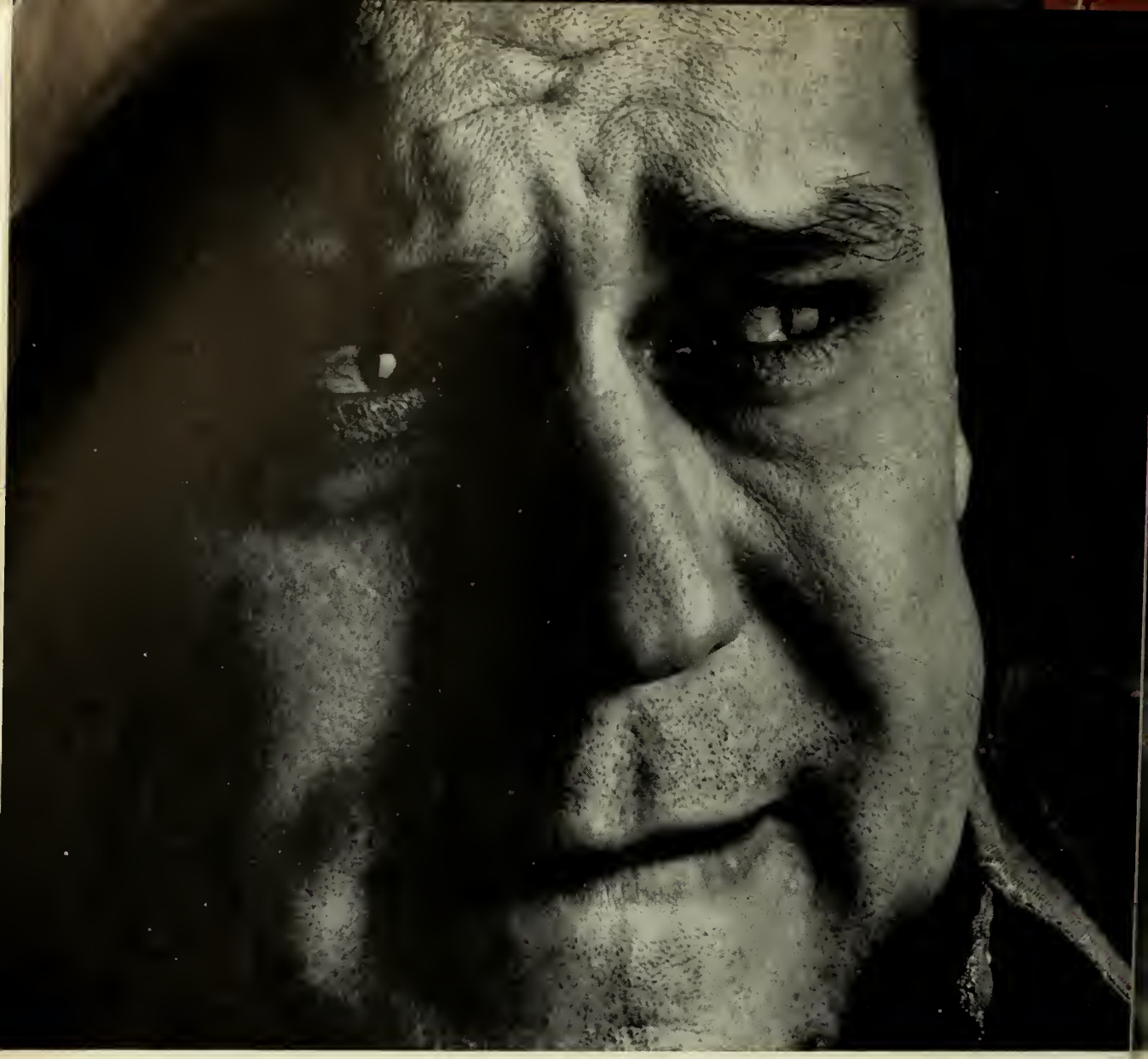


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Precautions: In suspected staphylococcal infections, perform proper laboratory studies including sensitivity tests. If overgrowth of nonsusceptible organisms occurs (constant observation is essential), discontinue penicillin and take appropriate measures. Whenever allergic reactions occur, withdraw penicillin unless condition being treated is considered life threatening and amenable only to penicillin. Penicillin may delay or prevent appearance of primary syphilitic lesions. Gonorrhea patients suspected of concurrent syphilis should be tested serologically for at least 3 months. When lesions of primary syphilis are suspected, dark-field examination should precede use of penicillin. Treat beta-hemolytic streptococcal infections with full therapeutic dosage for at least 10 days to prevent rheumatic fever or glomerulonephritis. In staphylococcal infections, perform surgery as indicated.

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Incidence of Rheumatic Fever

Summary of an Eight Year Study of Incoming Freshmen at the University of North Dakota

EDWIN G. OLMSTEAD, M.D., and JANE CHURCHILL

Grand Forks

In the fall of 1958 a four-year study was undertaken to determine incidence of previous rheumatic fever in incoming freshmen to the University of North Dakota.[1] The scope of the study included the incidence of rheumatic fever, incidence of rheumatic valvular heart disease, the number of students with a history of rheumatic fever who were on prophylaxis against Group A-Streptococcus, and the number of recurrences of rheumatic fever in students on prophylaxis as compared to those who were not. This study was published in the *Journal-Lancet* in June 1962.

It was decided to continue the study another four years, concluding in December 1965. It is now possible to give a summary of the total eight-year study.

Briefly, the University of North Dakota is a state supported institution with a freshmen enrollment of about 1,200 students. The student body is drawn largely from North Dakota and northwestern Minnesota and has a ratio of men to women of about 3:1 in the freshmen class. Incoming students are required to fill out a health form and have a physical examination by their home physician, usually in the summer preceding entrance.

In the present study, as in the previous study, all students with a history of rheumatic fever as noted on the health form were called to the student health center where a complete history and physical examination were carried out. Chest X-rays and electrocardiograms were obtained on all students with valvular heart disease, and where necessary other diagnostic measures were included. A few students were not counted in the survey as the past history of the rheumatic attack was not considered adequate to justify the diagnosis of acute rheumatic fever.

During the second four years of the study, the university had the services of a full-time director of student health who referred patients for evaluation of cardiac murmurs heard during examinations for noncardiac related complaints. Some of these patients proved to have rheumatic heart disease and were included in the study.

Figure 1 shows the percentage of entering freshmen having a history of rheumatic fever for the years 1958-1965 inclusive. It should be noted that over this period of years there has been little change in the number giving a history of having had rheumatic fever. About 2% give a history of having had a bout of rheumatic fever in the past.

Figure 2 shows the percent of freshmen with

EDWIN G. OLMSTEAD *is associate professor of medicine and* JANE CHURCHILL *was a Lederle Medical Student Research Fellow, University of North Dakota, Grand Forks.*

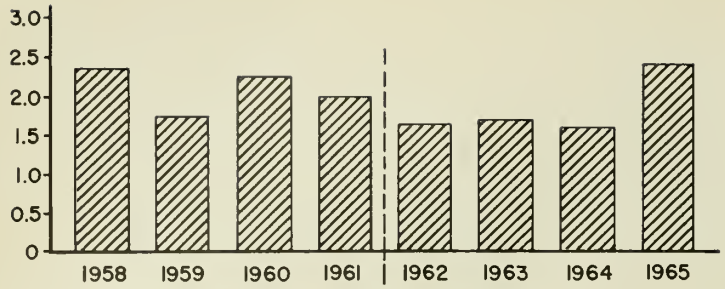


Fig. 1. Percentage of entering freshmen at University of North Dakota giving a history of having had rheumatic fever, 1958-1965. Broken line indicates division between 2 periods of study.

a history of rheumatic fever found to have a valvular heart disease. Previous study had indicated a precipitous drop in the incidence of valvular heart disease among freshmen having a history of rheumatic fever for the years 1958 to 1961 inclusive. This leveled off during the years 1962 to 1965 and demonstrated that approximately 20% of freshmen who gave a history of rheumatic fever before entering the University were found to have valvular heart disease. This represents a considerable decline from the years 1958 to 1959 when over half of the incoming freshmen with a history of rheumatic fever were found to have valvular heart disease. It should again be emphasized that this drop in the percentage took place even though the number of students entering with a history of rheumatic fever stayed relatively the same over the eight-year period.

SUMMARY OF FOUR-YEAR STUDY OF FRESHMEN STUDENTS WITH HISTORY OF RHEUMATIC FEVER 1962 TO 1965 INCLUSIVE

	Number of freshmen	*Percent of total number of freshmen*	*Percent of freshmen with history of rheumatic fever*
	(4606)		
Total freshmen	5426		
Total with history of rheumatic fever	(99) 103	(2.2) 1.9	100
Total with valvular heart disease	(35) 23	(0.8) 0.4	(35.3) 22.4
Total with history of rheumatic fever on prophylaxis	(20) 30		(20.2) 29.0

Figure 3 shows the percent of entering freshmen with a history of rheumatic fever on prophylaxis. The previous study from 1958 to 1961 showed that in 1961 all incoming freshmen students with a history of rheumatic fever were on prophylaxis. This represented a very small number of students and subsequent study showed this to be an error. Recheck of the data for the year 1961 showed the number to be only about 40%.

It will be noted from Figure 3 that the number of incoming freshmen students on prophylaxis has not increased to any appreciable degree in the second half of the study as compared with the first half. Generally about 35% of students with a history of rheumatic fever are on prophylaxis when they arrive.

Figure 4 shows the percentage of freshmen having valvular defects on prophylaxis at the time they enter. Interestingly enough, the second half of the study shows fewer patients having valvular defects on prophylaxis when they came to the university than was the case in the first four years of the study. This will be discussed later.

The Table is a summary of the second four-year study of freshmen students with a history of rheumatic fever. The numbers in parentheses indicate the same parameters as used to measure the first survey. As would be expected from the study of the graphs, the total number during these two periods did not appreciably change, being about 2.2% of the first entering class and 1.9% of the second entering class. The total having valvular heart disease, however,

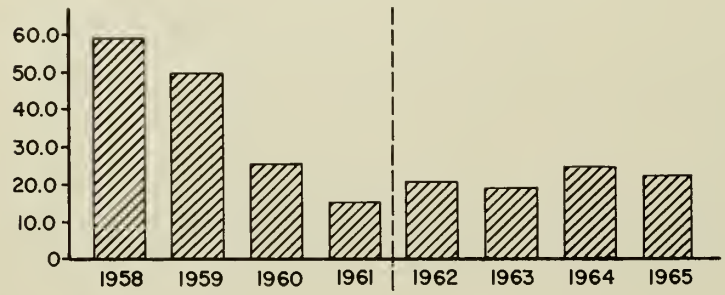


Fig. 2. Percentage of entering freshmen with a history of rheumatic fever found to have valvular heart disease, 1958-1965. Broken line indicates division between 2 periods of study.

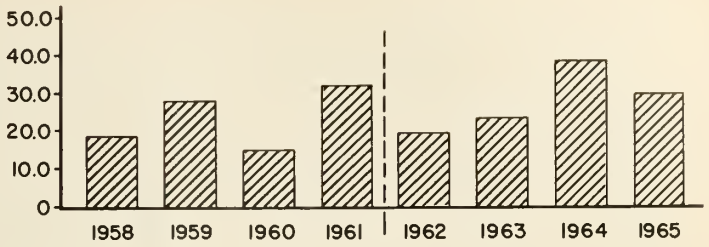


Fig. 3. Percentage of entering freshmen with a history of rheumatic fever who were on prophylaxis, 1958-1965. Broken line indicates division between 2 periods of study.

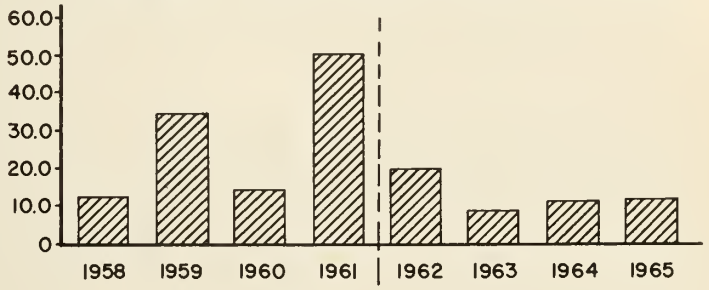


Fig. 4. Percentage of entering freshmen with history of rheumatic fever and valvular heart disease on prophylaxis, 1958-1965. Broken line indicates division between 2 periods of study.

declined significantly from 35% of those with a history of rheumatic fever having valvular heart disease in the first study to 22.4% of those with a history of rheumatic fever having valvular heart disease in the second study. There was a slight increase of patients on prophylaxis from 20.2% of the first group to 29% of the second group.

In October 1961 a low-cost prophylaxis program against rheumatic fever was initiated by the state of North Dakota in cooperation with the state medical society and the state pharmaceutical association. This is a prophylaxis program similar to those of other states where either intramuscular or oral penicillin or sulfadiazine is provided at cost for patients in order to prevent recurrences. In December 1965 the total number of registered patients in this program was 1,637.

Figure 5 shows a cumulative graph of the registrations for prophylaxis by six-month intervals from 1961 to 1965. It is of interest to note that the slope of the graph is largely unchanged during this period. This would suggest that the rate at which the prophylaxis program has been utilized by the physicians in the state has not changed over the four-year period. In other words, physicians who put their patients on prophylaxis in 1961-1962 continued to do so in 1964 to 1965; however, physicians who did not put their patients on prophylaxis during 1961 to 1962 probably did not put them on in 1964 to 1965 to any appreciable degree. If more physicians would have utilized the program as it developed, one would have expected that the cumulative graph would have shown a sharp upturn at some point rather than a constant slope. Some caution is in order in interpreting the above statistics. For instance, during the second half of the study there were only a total of 23 patients with valvular heart disease in the four-year period of study, or an average of only about 6 patients per year. If only 3 of these were on prophylaxis at the time of coming to the university, this would indicate that 50% were not on prophylaxis—but, again, this represents only 3 patients. In this sense, the use of percentages can be misleading without attention to the actual number of patients involved. It is difficult to state why the prophylaxis program is

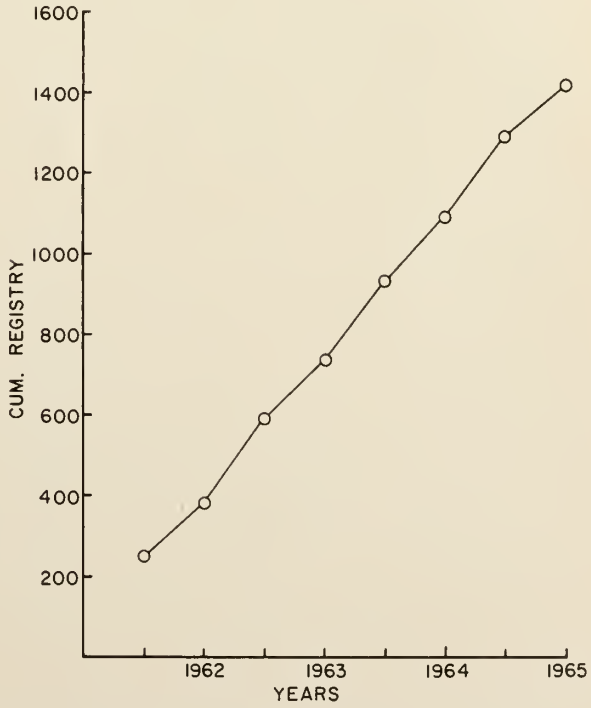


Fig. 5. Cumulative graph of numbers of registrants for state prophylaxis program 1961-1965.

not utilized to a greater extent. One possibility is that the criteria for the diagnosis of rheumatic fever are not as sharp as formerly. In some instances now it is difficult to be positive whether a child with a generalized arthralgia and a few other findings actually has rheumatic fever or not. There is a natural reluctance to condemn the patient to many years of prophylaxis if it is not necessary. On the other hand, this hardly explains the low number of freshmen with obvious valvular heart disease who are not on prophylaxis.

In summary, this represents an eight-year study of rheumatic fever in incoming freshmen at the University of North Dakota. The incidence of a history of rheumatic fever has not changed appreciably over this eight-year period and amounts to approximately 2%. The over-all incidence of rheumatic valvular heart disease, on the other hand, has dropped markedly during this period, reaching a low level in 1961 and continuing until the present time. Approximately 20% of all freshmen who give a history of rheumatic fever are found to have valvular heart disease upon entering the University. Although the state prophylaxis program has been available now for approximately five years, there is no indication from this study that the program has resulted in an increase in the number of students placed on rheumatic fever prophylaxis. As a matter of fact, there were fewer freshmen with a history of rheumatic fever having valvular heart disease on prophylaxis when they came to the university, during the second half of the study, 1962 to 1965, than there was during the first half of the study, 1958 to 1961, when the state prophylaxis program was not in use.

The author wishes to thank Dr. R. C. Eelkema, director of the student health center and Miss Ruth Noren of the nursing staff of the student health center for their kind cooperation in assisting in the above investigation.

This paper was presented at the meeting of the North Dakota Medical Society in Fargo, April 29, 1966.

REFERENCE

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Results from Shunting Procedures for Internal Hydrocephalus

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THE MAGNIFICENT ANATOMIC and physiologic studies by Dandy and Blackfan[1] clearly define the basic pathology of internal hydrocephalus. After correlating the studies of their predecessors, conducting carefully planned and meticulously executed experiments on animals, studying autopsy specimens, and observing human patients with internal hydrocephalus, they were able to reach the following conclusions which still provide a basis for our understanding of internal hydrocephalus today:

1] That the cerebrospinal fluid is formed in the ventricles faster than it can be absorbed and that the aqueduct of Sylvius is essential for its escape

2] That cerebrospinal fluid is derived mainly from the choroid plexuses, probably both by filtration and by secretion

3] That an increase of cerebrospinal fluid is caused by general venous congestion of the brain and that this increase of fluid ceases when the congestion is relieved by collateral circulation

4] That there is a definite impermeability of the fluid-forming structures

5] That there is a rapid and constant formation and absorption of cerebrospinal fluid and that a new supply is formed and absorbed at least every four to six hours

6] That cerebrospinal fluid is absorbed directly into the blood and that absorption is from the entire subarachnoid space

7] That there is practically no absorption from the ventricles

8] That maintenance of an equilibrium between the formation and absorption of the cerebrospinal fluid necessitates a communication between the ventricles and the subarachnoid space

9] That this communication is solely by the foramina of Magendie and Luschka and that there are no valves at these openings

10] That internal hydrocephalus can be divided into two anatomically different types, depending on the patency or occlusion of communication between the ventricles and the subarachnoid space.

Dandy went on to elucidate the principles for surgical treatment. He observed that, in the obstructive type of hydrocephalus, the obstruction should be removed, while in the communicating type of internal hydrocephalus, it is necessary to increase the area for absorption of fluid. He pointed out that if, in the obstructive type, the obstruction was at the foramina of Magendie and Luschka, this obstruction could be relieved surgically. But if the occlusion was located at the aqueduct of Sylvius, the problem of making an opening was obviously more difficult. He further noted that, before undertaking an operation to relieve an obstruction of the aqueduct of Sylvius, it would be necessary to determine the amount of absorption from the subarachnoid space. If a low subarachnoid absorption was present, relief of the obstruction would merely transform an internal hydrocephalus of the obstructive type into one of the communicating type.

Dandy suggested that the rational treatment in the communicating type of hydrocephalus was to drain the fluid into other tissues where adequate absorption existed. He then devised three physiologically sound, but technically difficult, operative procedures for internal hydrocephalus: [1] For the obstructive type in which adequate absorption from the subarachnoid space was present, he recommended third ventriculostomy with drainage of the cerebrospinal fluid into the cistern interpeduncularis. [2] For obstructive hydrocephalus from incomplete stenosis of the aqueduct of Sylvius but with adequate absorption from the subarachnoid space, he recommended drainage through a tube placed in the aqueduct of Sylvius. [3] For the communicating type of hydrocephalus, he recommended choroid plexectomy in an attempt to reduce the amount of formation of cerebrospinal fluid to

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the point where it could be properly absorbed.

The technical complexity of these operations, the attendant morbidity and mortality from them, and the high percentage of cases of obstructive hydrocephalus without adequate absorption in the subarachnoid space provided the impetus to develop other procedures to handle the problems.

The first was an operation developed by Torkildsen[2] in 1939. He used a rubber catheter to shunt cerebrospinal fluid from the lateral ventricle into the cisterna magna in cases of obstructive hydrocephalus from inoperable occlusion of the aqueduct of Sylvius. Time proved this operation to be a highly successful method of bypassing an obstructed Sylvian aqueduct with a low rate of mortality and morbidity.

The introduction of plastic materials, well tolerated in the human body, then led to the development of additional shunting procedures with plastic tubes for the various types of internal hydrocephalus. The first major advance in this direction was made by Matson,[3] when he devised an operation for communicating hydrocephalus that shunted cerebrospinal fluid from the lumbar subarachnoid space through a plastic tube into a ureter. Shortly thereafter, Nosik[4] suggested shunting cerebrospinal fluid from the temporal horn of the lateral ventricle into the mastoid antrum. In 1952, Nulsen and Spitz[5] described a method of shunting cerebrospinal fluid from the ventricle to the jugular vein. Five years later, Pudenz and his associates[6] described a technique for shunting cerebrospinal fluid into the right auricle of the heart from a lateral ventricle. The relative technical simplicity of these shunting procedures, the low operative mortality and morbidity from them, and the excellent immediate postoperative result in the control of the hydrocephalus from them encouraged us to use shunting procedures in the treatment of this disease. The immediate good results, however, belie the long-term results; long-term complications are the rule rather than the exception.

RESULTS

Results of our experience (1951-65) with 4 different shunting procedures are shown in the tables.

Of the 90 procedures carried out on 85 patients (Table 1), 57 were on patients under 3 months of age; 14, on patients 3 to 6 months old; 2, on patients aged 6 months to 1 year; 4, on patients 1 to 2 years old; and 13, on patients over age 2.

Table 2 gives the survival rate from the shunting procedures. It should be noted that all shunts to the ureter were carried out between the years of 1951 and 1958, and the shunts to the fallopian tube were done during the years of 1956 and 1957. Since 1958, ventriculoatrial shunts utilizing the Pudenz-Heyer valve and, more recently, their flushing device have been used exclusively for both types of internal hydrocephalus.

Table 3 is an analysis of the time interval between the first shunting procedure and death. It should be noted that the greatest loss occurs within the first two years after the shunting procedure and that, if the patient survives two years after the shunting procedure and if his hydrocephalus is well controlled, it would seem that his outlook for continued survival seems reasonably good. The single death in the more-than-four-year group was a child who obstructed his shunt to the ureter seven years after the original operation. A ventriculoatrial shunt was then done, but he developed endocarditis and eventually died from the renal failure of chronic glomerulonephritis.

Table 4 shows the causes of death as related to the different types of shunting procedures. It should be noted that infection has been the major cause, accounting for half of all deaths and manifesting itself in meningitis, endoearditis with septicemia, and pneumonia. It should further be noted that failure of the shunt accounted for almost a third of the deaths. A multitude of technical difficulties accounts for the failure of these shunts. The most frequent cause of failure in shunts to the ureter is obstruction of the spinal end of the shunt by an overgrowth of arachnoid occluding it. In fact, it is likely that, within five to ten years after this shunt is performed, it becomes occluded at the spinal end. In the shunts to the fallopian tube, however, failure of the shunt usually occurs from obstruction in the peritoneal end, where the fallopian tube overgrows and occludes the end of the plastic tube.

Failure of the ventriculoatrial shunts occurs principally for two reasons: [1] If, in the infant, the valve in the right auricle gradually ascends from the auricle into the inferior vena cava, a fibrous sheath from the vena cava proliferates over it and obstructs it. It is, therefore,

TABLE 1

SURGICAL MATERIAL

	Shunts to ureter	*Shunts to fallopian tube*	*Shunts to mastoid*	*Ventriculo-atrial shunts*	*Total*
Male	14			32	46
Female	13	6	4	21	44
Total	27	6	4	53	90

TABLE 2

SURVIVAL FROM SHUNTING PROCEDURES

	Under 1 year	*1 to 2 years*	*2 to 3 years*	*3 to 4 years*	*4 to 5 years*	*More than 5 years*	*Total*	*Percent*
To ureter						13	13	48%
To fallopian tube						3	3	50%
To mastoid							0	
VA shunts	5	4	3	5	5	14	36	68%
Total	5	4	3	5	5	30	52	58%

TABLE 3

DEATH FOLLOWING SHUNTING PROCEDURES

	Under 1 month	*1 to 3 months*	*3 months to 1 year*	*1 to 2 years*	*2 to 4 years*	*More than 4 years*	*Total*
To ureter	1	5	3	3	1	1	14
To fallopian tube		1	2				3
To mastoid	1	2		1			4
VA shunts	1	4	3	5	3	1	17
Total	3	12	8	9	4	2	38

TABLE 4

CAUSES OF DEATH

	Shunt failure	*Meningitis*	*Electrolytic imbalance*	*Brain tumor*	*Renal failure*	*Endocarditis with septicemia*	*Pneumonia*	*Surgical*	*Posterior fossa cyst*	*Total*
To ureter	4	5	1	1	1	1	1			14
To fallopian tube	3									3
To mastoid	1	3								4
VA shunts	3			1	1	5	4	2	1	17
Total	11	8	1	2	2	6	5	2	1	38

necessary to revise these shunts frequently during the period of rapid growth in the first two years of life, advancing the valve into the auricle whenever X-rays reveal it to be near the junction of the auricle with the superior vena cava. [2] In the other instance, the ventriculoatrial shunting tube in the ventricle becomes obstructed from a proliferation of choroid plexus or ependyma into the shunting tube. This complication occurs less frequently if the tube in the lateral ventricle is advanced into the anterior horn of the lateral ventricle so that the side holes lie anterior to the foramen of Monro, not in contact with choroid plexus of the lateral ventricle.

It should be noted that we lost only one child from electrolyte imbalance following a shunt to the ureter. This was largely due to the fact we were alerted to this complication and were able to take prophylactic measures against it.

The patient who died from a brain tumor following a shunt to the ureter was an interesting case. The brain tumor proved to be a papilloma of the choroid plexus which was not visualized by the air study carried out preoperatively

TABLE 5

SHUNT REVISIONS NEEDED ON SURVIVALS

	0	*1*	*2*	*3*	*More than 3*	*Different type of shunt*
To ureter	6	3				4
To fallopian tube	2		1			
VA shunts	15	9	7	4	1	
Total	23	12	8	4	1	4

but which undoubtedly was the basis for this child's communicating hydrocephalus.

Of the 2 operative deaths, 1 was from perforation of the internal jugular vein in the chest during revision of a ventriculoatrial shunt and the other was from pontine hemorrhage following too rapid decompression of the hydrocephalus. The patient who died from a brain tumor in the group of ventriculoatrial shunts was a case in which the shunt was done as part of the treatment for an inoperable brain tumor. In the patient who died from a posterior fossa cyst, the ventriculoatrial shunt had provided excellent de-

TABLE 6

PHYSICAL AND MENTAL STATUS OF SURVIVAL CASES

	Normal mental and physical development	*Normal mental; moderate physical disability*	*Normal mental; severe physical disability*	*Retarded but educable; normal physical development*	*Retarded but educable; moderate physical disability*	*Retarded but educable; severe physical disability*	*Uneducable; severe physical disability*
To ureter	6	1	1	3	1		1
To fallopian tube	1	1			1		
VA shunts	19	2	5	4	3	3	
Total	26	4	6	7	5	3	1

compression of the lateral ventricles and of the third ventricle. But the enormous cystic mass in the midline of the posterior fossa filled with cerebrospinal fluid apparently derived its source of fluid from the choroid plexus of the fourth ventricle, and communication with the subarachnoid space and fourth ventricle was obstructed.

Table 5 shows the number of shunt revisions that have been needed on the patients who survived the various types of shunting procedures. It should be noted that 4 of the patients having shunts into the ureter subsequently required ventriculoatrial shunts at a later time. The remaining 9 survivors are probably shunt-independent and have made an adjustment to their hydrocephalus. Of those requiring ventriculoatrial shunts, one was eleven years after the original shunt to the ureter and the others were eight, seven, and six years after the original shunt. Of the 15 patients with ventriculoatrial shunts requiring no revisions, 6 are less than 2 years postoperatively and may need a shunt revision to compensate for their continuing physical growth. The one patient requiring more than 3 revisions has had 7 revisions of his shunt.

Table 6 is an analysis of the physical and mental status of the surviving patients. Although some of these patients have had psychological tests to determine their intellectual capabilities, in general the evaluation of mental status has been on an empirical basis during recheck examinations. It should be further noted that, of the total group of 90 shunting procedures, 24 were carried out on patients who had survived plastic repair of a meningocele or myelomeningocele and had developed internal hydrocephalus. Of these 24 patients, 11 died and 13 survived. Of the 13 survivors, 6 fall in the group with normal mental development but severe physical disability—a paraplegia resulting from a myelomeningocele.

CONCLUSIONS

With improvement in techniques, careful selection of cases, expanded use of prophylactic antibiotics, and judicious revision of the shunts as indicated, the continued use of ventriculoatrial shunts in the treatment of internal hydrocephalus would seem to be indicated. The results obtained thus far from the use of this procedure seem to compare favorably with results obtained from more physiologic intracranial operations.

SUMMARY

Ninety examples of 4 different types of shunting procedures carried out on 85 patients are presented. The follow-up periods varied from one to fifteen years, and the survival rate was 58%. The critical period for survival occurred between the first month and the second year following the shunting procedures. The most common cause of death was infection, and about one-third of the deaths resulted from failure of the shunt to function properly. Our experiences suggest that about one-third of patients with communicating hydrocephalus eventually adjust to their hydrocephalus and become shunt-independent. Ventriculoatrial shunts carried out for internal hydrocephalus in infants and small children are likely to need revisions and need frequent and careful follow-up observation. Fifty percent of the cases surviving a successful shunting procedure showed normal mental and physical development. About 70 percent showed apparently normal mental development.

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Functional Problems Confronting the Obstetrician

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PHYSICIANS WHO PRACTICE obstetrics are in a remarkable position to help their pregnant patients with their emotional problems if advantage is taken of the unique doctor-patient relationship that develops during the course of pregnancy. The type and strength of this relationship is well demonstrated in that few women forget who delivered them, while many tend to forget who attended them for other medical problems.

Although pregnant women may present with unusual specific problems, most complain of common symptoms such as abdominal discomfort, nausea and vomiting, urinary frequency, headaches, backaches, weight gain, insomnia, and depression. For effective treatment the physician must recognize that many of these patients with "female trouble" are really "troubled females" and, therefore, he should not be concerned only with the "physical aspects" of the complaints. In managing these various and sundry complaints properly and without delay, the determination of whether they are emotional in origin must be determined early on the basis of the presence of positive findings—not only by exclusion. A diagnosis so made will be convincing to the doctor as well as to the patient. A positive diagnosis results primarily from the history of the present illness and its precipitating factors, combined with background information about the patient and her family, and evaluating the patient's facial expression, attitude, and concern about her symptoms.

In addition to these common complaints, patients may consult their obstetricians about more unusual problems, such as request for an abortion, habitual abortion, pseudocyesis, infertility, frigidity, nonconsummation of marriage, pruritus, and peculiar behavior of their husband—including the adamant demand that he be present at each routine examination and at the time of delivery.

Each of these complaints need not be considered individually, since general principles of management are applicable to all. It is important that the doctor neither be frightened of nor overconcerned about symptoms. What is behind the symptom is important. The doctor should realize that all emotional symptoms are handled at least in part by psychotherapy, which can best be defined as the doctor's manipulation of the doctor-patient relationship in such a manner that the patient's condition improves. Properly conducted psychotherapy proceeds differently in different categories of illness. In addition to considerable self-understanding on the part of the physician, it requires a broad medical knowledge, considerable experience in listening, understanding, interpreting, and knowing when reassurance is indicated.

Emotional symptoms of greater or lesser degree occur with every pregnancy. The patient consults the doctor, and a psychotherapeutic relationship has begun. Everything the doctor does should be for the benefit of his patients. Selzer[1] states anything the doctor does that fails to meet this criterion has no place in patient management. Addressing the pregnant woman by her first name is a common practice. Since this practice does not specifically benefit the patient, and may be detrimental, it should not be used routinely. This familiarity unduly increases dependency problems and encourages child-like behavior in the patient and thus does not assist in the formation of a mature patient-doctor relationship. The doctor-patient relationship is not a "friendly" relationship. When the patient-doctor relationship is used correctly by the obstetrician, many conditions—ranging from the usual, such as abdominal discomfort, to the unusual, like habitual abortion—can be treated effectively. Some of these, doctors have treated successfully

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in the past but have credited medications and procedures for their therapeutic triumphs, rather than the patient-doctor relationship.

According to Tupper and Weil,[2] supportive psychotherapy is the major therapeutic factor in the management of habitual abortions and the prevention of pregnancy losses. They believe the common denominator of all treatment methods of habitual abortion is the doctor's conveyed belief in the patient's capacity to continue to term. They found no evidence that deficiency in diet, lack of hormone support, trauma, or travel play any role in causation. Their studies indicate that women who habitually abort are either (1) basically immature women who cannot accept the responsibility of mature femininity, namely, motherhood, or (2) independent, frustrated women who have been conditioned to yearn for the rewards of the male world and feel that the role of being a mother is unsatisfying. These women tend to abort when subjected to extremely stressful circumstances, which include an absent or uninterested husband, or a disinterested doctor. These women appear to abort when their pregnancy is not accompanied by the environmental support and prestige they feel entitled to.

These physicians utilized supportive interviews with relatively few limits; they even allowed their patients to contact them at any time by telephone. With this method of treatment they obtained 16 full-term births in their 19 supportively treated patients, while in the control group of 19, there were only 5 full-term births.

It is not apparent from Tupper and Weil's[2] paper if a determination was made whether or not these women were psychiatrically stable enough to raise children effectively, or what happened to the mothers after delivery. That this should be done is well brought out by a recent paper presented at the American Psychosomatic Society by Dr. Van den Bergh and associates.[3] These doctors studied 9 habitual aborters who had Shirodkar operations and found that 8 of these patients had either a postpartum psychosis within six months of delivery, or required psychotherapy during pregnancy. He stated, "Such a procedure may force the habitual aborter into unwanted motherhood and may bring on a psychosis through the sharpening of her underlying emotional conflicts." If this work is substantiated by others, the enthusiasm some have for this operative treatment will diminish.

The treatment of infertility is a challenging aspect of obstetrics, yet until recently, only a few physicians have seriously considered the significance of emotions as a factor in sterility, although primitive people successfully used incantation and religious rituals to alleviate the sterile state. It is obvious that psychotherapy is important in the alleviation of infertility because "successes" occur in many cases following such simple treatment modalities as sedation, vacation, adoption, passing a uterine sound, or just beginning an infertility investigation.

Finding and treating a minor defect in an infertile patient and neglecting to study her personality is similar to looking in the mouth of a frightened, anxious woman, noting that it is very dry, and prescribing a mouthwash. Observed pathology may not be the cause of the patient's problem. The diagnosis of psychogenic infertility is usually made on a negative basis, although there are many reports in the literature of the personality pattern of the infertile woman. At times the doctor should suspect the diagnosis.

Most who study the personalities of sterile women find in them a chronic failure to adapt to the demands of a mature interpersonal relationship, not only in the sexual sphere, but in every phase of reality. Mandy[4] states that infertile women can be divided into 1 of 2 types: either typically feminine and juvenile, or aggressive and masculinely competitive with a wish to dominate the entire household. Is this the kind of person who should have a child? Infertile couples should be studied as thoroughly as prospective adoptive couples, and the doctor must not take each request for a baby as a command and a test of his ability. Some couples simply should not have a pregnancy because of emotional maladjustment. In one case of infertility caused by infrequent intercourse, the fear of referral to a psychiatrist produced more frequent coitus, which resulted in conception. This couple, now with 2 children, is in the process of divorce. After studying this couple, it is obvious they never should have had children. The husband is a passive, feminine man who would rather be "with the boys" while his wife is a "driving" woman with masculine interests.

An extremely interesting problem which occasionally presents in an infertility study is the nonconsummated marriage. Sandler[5] reports "Five percent of all women attending an infertility clinic are virgins, intercourse taking place between the thighs, and neither party being aware of the absence of penetration." In many of these cases that have been studied and treated, the male has been found to be an active or latent homosexual. When this information has been clarified some of the couples have gone back to their mutual masturbation or "brother and sister" relationship and life for them is again smooth. In a fascinating case of this type in which the

husband is currently under active psychiatric treatment, the reason for his inability to perform has been determined. Just as he is about to penetrate, the thought enters his mind that he is having intercourse with his mother, and concomitantly his erection disappears. His mother had slept with him and fondled his genitalia until he was 16, when he decided to stop this behavior. His mother was not involved in this manner with his twin brother, who is a normal heterosexual male. Recently this couple adopted a child. Now the marriage is really disturbed.

Postpartum psychosis or postpartum depression is a serious problem for the obstetrician. Because it is not a specific entity and simply a psychiatric illness whose precipitating factor is this particular pregnancy, it should be treated like any other depression precipitated by an unhappy marriage, death in the family, financial reverses, and such. Noyes[6] states, "Mental disorder may be associated with pregnancy or puerperium. There are, however, no psychoses specific to either of these periods. Both are periods accompanied by physiological and psychological stress. Latent or repressed psychological material may, under the stress of maintaining physiological homeostasis and of the emotionally significant situation, prove too great for the patient's ego resources with the result that psychopathological reactions occur."

It is impossible to predict who may and who may not develop a postpartum psychosis. Zillboorg[7] states "Pregnancies that are marked by psychological tensions, severe moods, and anxieties are not really abnormal pregnancies, and so-called common sense to the contrary, the so-called psychologically uneventful, serene, and sunny pregnancies are apt to be harbingers of severe and even malignant postpartum reactions. This does not mean that all psychologically uneventful pregnancies are forerunners of postpartum psychoses, but they frequently may be, whereas the so-called psychologically stormy pregnancies seldom are such forerunners." Martin[8] reports that after the first puerperal psychosis, there is only a 20% chance that the patient has a recurrence with the next pregnancy. From this it is clear that history of postpartum psychosis is no indication for therapeutic abortion.

Since there are few acceptable medical indications for therapeutic abortions today, more patients will be presenting to the obstetricians with a request for therapeutic abortion because of depressive feelings and suicidal gestures. Bernstein[9] concludes there are no valid psychiatric indications for therapeutic abortion because suicide is a rare concomitant of pregnancy (Barno[10] reports only 14 in Minnesota in approximately 1.5 million births since the institution of the Minnesota Maternal Mortality Study) and because adequate treatment methods for depression are available, besides the fact therapeutic abortion has its own psychiatric morbidity.

An obstetrician not only can be helpful in arranging psychiatric care for his pregnant patient who needs it, but he can be instrumental in obtaining psychiatric treatment for her spouse. If a patient's husband demands that he be present in the delivery and examining room, or both, the physician should interpret this as evidence of paranoid-tinged thinking until proved otherwise. This knowledge by the obstetrician has resulted in the diagnosis of a number of cases of paranoid schizophrenia among husbands of patients receiving obstetrical care at the University of Minnesota Hospitals who were subsequently hospitalized for psychiatric care.

Although fairly good results are obtained from the treatment of emotional problems of the obstetric patient, prevention should be the goal and the obstetrician is in an excellent position to contribute. He is fortunate in having a young, usually motivated patient periodically, who feels he is not only her doctor, but her teacher, counsellor, confidant, and psychiatrist. The obstetrician with his limited training in psychotherapeutic techniques may not be able to change his patient basically, but he should be able to help his patient raise her child more effectively. He should emphasize the importance of giving her newborn child love, security, and acceptance and should avoid subjecting the child to fear, isolation, unnecessary pain, and anxiety. He should also impress upon the mother to raise "the girl as a girl" and inculcate in her healthy attitudes on anatomy, physiology, and sex. If this is done, the daughter will be better equipped to handle all her future problems, including pregnancy.

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Surgery—Yesterday and Tomorrow

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THE POWER OF APPOINTMENT

This great occasion* signalizes the importance of the Power of Appointment. The veterans hospitals, as did this one, came into being in 1927 to provide hospitalization and continuing medical care for veterans of World War I. Official institutional affiliation with universities was unheard of in those days. However, several early appointees on this hospital staff are members of the university's department of surgery and did much to provide superior surgical care for veterans on these premises. Among these, I need only mention Stanley R. Maxeiner, Thomas J. Kinsella, and Leo C. Culligan.

Toward the end of World War II, Dr. Paul Magnuson of Chicago, a native of St. Paul, together with the late Paul Hawley, for many years director of the American College of Surgeons, conceived the idea of a new type of cooperative management in which veterans hospitals would be operated under the aegis of university deans' committees. It has been my privilege to serve as a member of such a committee and to view at first hand the problems involved in integrating the teaching and training activities of the hospital with those of the medical school.

We were very fortunate in our first appointment of John R. Paine as the university's representative as director of surgery. He, with colleagues who also had returned from active duty in World War II, did much to help create an academic atmosphere within the walls of this institution and set the basic pattern of the present university type of organization. When Dr. Paine left to assume the co-chairmanship of the department of surgery at the University of Buffalo, he was successively followed here by Drs. Lyle Hay, Donald Ferguson, William Kelly, and your present incumbent, Dr. Edward Humphrey, all of whom have helped build a departmental organization which reflects credit upon the medical school. Over the years this hospital with its superior staff and splendid laboratories has frequently been cited as one of the finest amongst the veterans hospitals of America.

In my view, the Power of Appointment is the most fundamental element to the success of any institutional enterprise. In many respects, it is tantamount to ownership. Moreover, that authority provides the opportunity to energize and lend direction to the growth of great enterprises. I argued this point unsuccessfully seventeen years age before the Board of Regents of our University with reference to placement of Mt. Sinai Hospital on the campus of the University of Minnesota. Yet that failure was a significant factor in directing notice to the great need of the medical school for additional hospital facilities, a circumstance which gave us a considerably larger Mayo Memorial Hospital than had been originally planned.

The idea of that native Minnesotan, Paul Magnuson, to place veterans hospitals under the jurisdiction of deans' committees has done much to ensure superior care and unique opportunities for professional training and research. It is especially appropriate that we signalize the importance of Dr. Magnuson's contribution today, inasmuch as some of his superiors failed to realize its potential in earlier years. Almost invariably, the success of major achievements constitutes triumph over the frustrations of inertia, indifference, and active opposition.

The Power of Appointment has made it possible for medical schools to lend a great forward thrust to medicine in this country. On another occasion, I cited the growth of veterans hospitals, under the supervision of deans' committees,[1] as 1 of the 3 great developments in American medicine in my time; the other 2 being the Flexner Report of 1910 and federal support of medical research under the aegis of the National Institutes of Health. In order to assure a continuing quality of academic excellence in performance in

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our hospitals, it is essential that pay schedules, equal to those of professional staffs at university medical centers, be established and maintained.

OBSERVED CHANGES ON SURGICAL WARDS OVER THE PAST FOUR DECADES

The medical student walking today's surgical wards is confronted with a sight quite different than that of my intern days in 1921 to 1922. More than one-third of the patients then admitted to Gillette Hospital in St. Paul came because of tuberculosis of lymph nodes and bone. Today none are to be seen there. A decade later, one of the important activities of our surgical service was treatment of pulmonary tuberculosis, first by thoracoplasty and later by lobectomy and segmental excision. These too have disappeared. Clinical medicine, in its strenuous wrestle with tuberculosis over decades, had far less to do with its virtual eradication in our country than did public health measures, such as pasteurization of milk and the slaughtering of tuberculous cattle. Eventual success came from 2 unexpected sources: microbiology gave us streptomycin through the genius of Dr. Waksman, and para-aminosalicylic acid came as a gift from pharmacology. On the global scene, the problem now is to eliminate tuberculosis in other areas of the world where it is still taking a large toll of human life.

Empyema following pneumonia was a frequent and challenging problem in my early years as a surgeon. Thanks to sulfonamides and penicillin, it has virtually been eliminated. Similarly, osteomyelitis has disappeared from our wards, a windfall from antibiotics.

Patients with exophthalmic goiter could always be seen on the wards. Iodized table salt has greatly reduced the prevalence of goiter and today the surgeon deals more frequently with cancer of the thyroid than with hyperthyroidism.

Appendicitis ranked amongst the first 10 causes of death in the United States until the late 30s. Then, through better knowledge of its origins and improved surgery and antibiotics, it tumbled to a low level in the mortality columns. The past 3 decades have witnessed a very significant reduction in the mortality of acute intestinal obstruction, through clarification of the consequences of obstruction, its clinical recognition and elaboration of nonoperative as well as surgical techniques of decompression.

The recognition of the conventional formal divisions of surgery, neurosurgery, orthopedics, and urology, came only in the late thirties. Before then, general surgeons covered the entire range of surgery, obviously doing only those operations well in which they had a special interest and experience.

Obviously, I have selected a few of the most startling contrasts confronting the student of my day who might look for those problems on the surgical wards today. I shall allude to only one other, that of infection. In my student days, the streptococcal cellulitic infections were the surgeon's chief dread; almost invariably these terminated in death, while staphylococcal abscess could be managed quite satisfactorily by surgical drainage. The chairman of our department of ophthalmology and otolaryngology, Dr. William Murray, lost an arm and then his life, in the space of a few days, because of a streptococcal infection. A brilliant young anatomy student, Lucretia Wilder, spilled a culture of streptococci on her forearm; a rabbit on the laboratory bench scratched her arm at the site of spillage and in a few days Dr. Wilder was gone.

SURVIVAL OF THERAPEUTIC PROCEDURES

Time is the sovereign arbiter of the survival of therapeutic agencies.

In driving from Zürich to Geneva, Switzerland, one bypasses a mountainous road which leads to the Leysin Peak. In the early winter months of 1928 I paid a visit to Rollier's Institution there, famous for use of heliotherapy for tuberculosis. Many a revisit is reminiscent of Wordsworth's Yarrow unvisited:

> "We have a vision of our own;
> Ah! why should we undo it?"

Rollier's establishment has disappeared almost from memory. In 1965 I was dismayed to find that the young people living in the area knew neither the name nor the place. Only that survives which continues to be useful.

The success of immunization for poliomyelitis is one of the great medical accomplishments of our time. The Metropolitan Life Insurance Company reports that in the decade 1953 to 1963, annual death benefits for poliomyelitis shrank from $653,000 to $7,000[2]—a decrease of almost 99%! What a boon to society if the threat of pyogenic infection could be eliminated like smallpox and poliomyelitis. Mastery of "infinite particulars,"[3] to use a phrase of the great poet and artist, William Blake (1799), will certainly someday solve and eliminate the hazards of hospital and surgical infections. It is of more than casual interest to us that Paré (1579) credited his success in wound management to "almost an infinite number of particulars."[4] No one appreciates more than the surgeon the importance of particulars in attainment of principles.

CONTRASTS IN ACTIVITIES OF SURGICAL HOUSE OFFICERS—THEN AND NOW

The surgeon of today would find it difficult to operate under the amateurish anesthesia available forty-five years ago. An intern, with perhaps some instruction from a surgeon or his intern predecessor on the service, or from an occasional consultant in anesthesia, gave the anesthetic—which not infrequently varied from premortal depth to a semiawake, struggling patient.

The present day house officer, with bottles of blood safely stored away for major procedures, is probably unaware that the only blood available for operation forty-five years ago was either in another patient's veins recuperating from an operation, or in a relative who was on hand, nearby, if needed. The hurry of the surgeon in operations was almost universal. The orientation of the clinician of that day in meeting fluid and electrolyte requirements reflects an abyss of ignorance.

There was no five-day week, no eight-hour day. Marriage amongst medical students was almost unheard of, as it was amongst interns. In fact, great clinicians like Osler and Halsted of the Hopkins considered marriage incompatible with the duties of a house officer.

There has been more than a 1,500% increase in hospital costs, and on many scores understandably so. An intern today casually orders laboratory tests on a patient, which, at current rates, would more than absorb the entire month's cost of hospitalization in 1920 with a $3.25 per diem. Medicine, in its broad aspects today, is a larger enterprise than the steel industry. The pursuit of health rightfully has become one of man's chief occupations and concerns.

Feeling more certain of his methods than his house officer analogue of forty-odd years ago, the budding clinician today exudes an air of confidence. Fortunately, he still reveals traces of love and sympathy for his fellowman by which important criterion all our patients judge us.

When I took over direction of the department of surgery, Jan. 1, 1930, one Surgical Fellow and 2 interns carried the responsibility for the care of 130 surgical patients. Today 15 interns and more than 100 Surgical Fellows discharge these functions for approximately 200 surgical beds. This very circumstance tells a great deal of the contrast in the care of patients then and now. Obviously, considerably more attention is lavished on each patient from the admitting department on through, including the laboratory, X-ray department, and other special technical aids. Occasionally some patients appear to have become irretrievably entangled in the intricate skeins and red tape of the admitting department. Last Sept. 6 marked the 65th anniversary of the attempt on President McKinley's life, which occurred at 4:06; at 5:22 p.m. he was on the operating table. In most hospitals today, even for emergencies, it takes longer for the patient to traverse the intricate maze of admitting procedures. Simplification and quickening of that process would be very acceptable to patients and physicians.

Intracardiac surgery had not been thought of. Organ transplantation was discussed by enterprising surgeons who appreciated only the technical difficulties, unaware of the great import of incompatibility and rejection of foreign tissue by the new host. One transferred blood, cornea, and fascia as today. Yet in 1932, Earl Padgett[5] had demonstrated that skin could only be transferred successfully between identical twins. The nature of the immunological barrier is still the main concern of ardent representatives of many medical disciplines pursuing this fascinating problem. It will not be an easy victory. When the answers come they will undoubtedly unlock the mysteries of a large number of disease entities.

INTERDISCIPLINARY ATTACK UPON PROBLEMS AND THE FUTURE

A heavy curtain of ignorance veils the future from our view. The oncoming generation is learning the great value of united interdisciplinary attack upon problems. History has shown how dependent advances in any medical field are upon discoveries in the biologic and physical sciences. Groups of like-minded specialists are not the best company for one another. To ideas generated in a weekly discussion seminar between the departments of surgery and physiology over the past thirty years can be attributed much of the credit for whatever my department has achieved. My advice to a young surgical chairman would be to find a Maurice Visscher in that basic discipline which flavor the activities of his department will reflect. Unfortunately they are too few. I am very thankful to have known one.

The lecture serves to emphasize what we know; the seminar what we do not know. In fact, future teaching as well as attack upon problems will certainly be characterized by multiple interdisciplinary seminar discussions with a critical use of information retrieval, employing the historical approach, in which the library and the historian will play increasingly important roles. The medical historian, schooled in a basic biologic discipline, with a keen interest in cur-

rent trends and developments, working in cooperation with representatives of biologic disciplines, can come, I believe, to play a pivotal role in attacks upon special problems. In areas in which there are extending and converging peninsulas of information, the medical historian and the library can catalyze effectively the important role of bringing isolated archipelagos of ideas into more meaningful arrangements and from which fusion enlightenment and understanding will come. The geography of land and ideas is not so dissimilar. Had interdisciplinary contacts been more frequent over the past century, microscopy and bacteriology would have had far greater meaning for the clinician. The lag periods between discovery and useful application of new-found knowledge have been far too long.

The past decade witnessed almost complete elimination of the threat of poliomyelitis. Perhaps through the agency of interdisciplinary research the next decades may show the way to eliminate rheumatic fever by the united efforts of microbiology, immunology, and pharmacology. The nature of the origin of some congenital deformities is being revealed by biochemistry, immunology, and pharmacology. Because of such advances, the cardiac surgeon of the twenty-first century will undoubtedly be less concerned with valve replacement and repair of congenital defects, while still concerning himself with management of coronary artery disease.

And what of cancer? In 1897, W. W. Keen,[6] then president of the American Medical Association, said the year 1947 would see the elimination of cancer. I was present at the first International Cancer Conference held in London when King George V's advisors read the king's reply[7] in which he said, "I note with interest that your object is research, both into the cure and causes of cancer. No doubt in the last resort the discovery of the cause is the only certain and absolute means of cure. But I am glad that you have not ignored the practical side of the problem. Remembering the thousands of sufferers from cancer, I feel that if your discussions lead to advance in diagnosis, treatment, or even palliation of the disease, this conference will have justified itself and earned the gratitude of mankind." King George has proved to be the best cancer prophet of this century.

Papanicoulau's program for early detection of cervical cancer succeeded in reducing mortality of uterine cancer by 50% in this country and stands out as the most important accomplishment in the cancer arena. The work of our own cancer detection center suggests that annual examinations and systematic efforts, widely applied, will make an important impact upon cancer of the rectum and breast. With refinement of precision roentgen techniques, cancer of the colon and stomach should also prove more amenable to surgical treatment. Early detection of lung cancer awaits elaboration of improved diagnostic techniques.

WHAT OF SURGICAL ACTIVITY AT CENTURY'S END?

What will the surgeon be doing in the year 2000? It is unlikely that appendicitis and gallstones will be eliminated. There are probably better prospects for preventing formation of some renal stones. Certainly the surgical management of peptic ulcer will be far less important than it is today. Simpler means will have been found of controlling gastric secretion. The surgeon 3 decades hence will surely have better techniques of hemostasis to ease his work. Pharmacologic aids will augment his ability to thwart and to contend with surgical infection. The agency of research in the whole sweep of surgery, from improved anesthesia to better wound management and postoperative care, will do much to simplify the surgeon's work and to improve the patient's lot.

The young surgical house officer of today, well accustomed to change, contemplating his own retirement in the first decade of the twenty-first century, will not be greatly surprised to find even greater emphasis lent research throughout the entire structure of medical training, though he may still be exposed to such empty phrases as "Give the medical school back to the students" and "Researchers are a dime a dozen." The climb up the academic ladder will be closed to those who eschew research.

While we acclaim with warm approval the significant victories of research of this generation, let us be mindful that achievement on many scores has exceeded the dreams of prophecy. In the decades ahead, interdisciplinary research will enable the oncoming generation to outstrip and overshadow contemporary performance. While we rejoice and exult over our successes, let us not fall victim to pride and self satisfaction exemplified in the vain prediction of one of surgery's immortals, Ambroise Paré, who said almost 400 years ago (1579) "Antiquity may seeme to have nothing wherein it may exceed us, beside the glory of invention, nor posterity anything left but a certaine small hope to adde some things, as it is easier to adde to former inventions."[8]

In concluding, I want to emphasize the important role of the university. Some of you who have sat in our surgical conference room have undoubtedly stayed to read John Masefield's

warm appreciation of the influenee of universities. Said the Poet Laureate (1946): "There are few things more enduring than a university. Religions may split into sects or heresies; dynasties may perish or be supplanted, but for century after century, the university will continue, and a stream of life will pass through it, and the thinker and the seeker will be bound together in the undying eause of bringing thought into the world."[9]

CONCLUSION

The eurtain falls on our yesterdays and there are gathered about us those who will roll up the eurtains of our tomorrows. While we await with high expeetations Heralds of the New Dawn, let us pause to pay tribute to this fruitful partnership of our Veterans Hospital and University and to the men of good-will who eonceived, nursed, and shaped the destiny of this great enterprise.

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Transactions of the North Dakota State Medical Association

EIGHTIETH ANNUAL MEETING

Bismarck, North Dakota, May 5, 6, and 7, 1967

(Continued from the August 1967 issue)

COUNCIL — SECOND SESSION

Sunday, May 1, 1966, Fargo, N.D.

Dr. Mahowald, Chairman of the Council, called the meeting to order at 5:00 p.m. Dr. Foster, Secretary, called the roll and the following responded: Doctors W. R. Fox, R. W. McLean, F. A. DeCesare, Dean Strinden, R. E. Mahowald, J. M. Van der Linde, G. Christianson, Keith Foster and R. S. Larson, new Councillor.

Others attending the meeting were: Doctors R. D. Nierling, V. J. Fischer, C. H. Peters, R. C. Painter, T. E. Pederson, Lee Christoferson, P. O. Dahl, J. J. Moses and Mr. Lyle A. Limond.

The Chairman of the Council read the "Duties of the Councillors" from the Constitution and By-Laws.

Mr. Limond brought up the subject of the letter from Mrs. Thakor that was tabled at the first session. Dr. Mahowald again read the letter, and after discussion, Dr. Foster moved that Mrs. Thakor be made aware of the booklet from the A.M.A., "Horizons Unlimited", and in addition, that the Council allocate, if the Treasurer states that this is favorable, the sum of $200.00 for the production of a booklet on Health Careers. Dr. Christianson seconded this motion. Dr. Strinden amended the motion to the effect that this sum of $200.00 be allocated with the stipulation that the Medical Education Committee be involved in editing this booklet and that this amount would not be turned over to their Health Careers Council until the total amount pledged from the other sources is available. Dr. Van der Linde seconded the amendment to the motion. Amendment to the motion was passed, and then the original motion was passed.

Dr. Nierling asked for an opinion or action on the auditing of the books. It was decided to postpone a decision until the December meeting of the Council.

Election of the Officers of the Council followed: Dr. Mahowald was elected unanimously as Chairman of the Council; Dr. Dean Strinden was elected unanimously as Vice-Chairman of the Council; and as Dr. Foster resigned his office of Secretary, Dr. Van der Linde was elected unanimously to the office of Secretary.

Dr. Dahl, new Speaker of the House of Delegates, brought up the business that was passed by the House of Delegates and the recommendations from the House to the Council; 1—An Honorarium for out-of-state speakers at the Scientific Session, to be considered and amount set at the discretion of the Council. After discussion, Dr. Foster moved that a sum of money up to $600.00 be made available to the Chairman of the Scientific Program for the purpose of providing an Honorarium for out-of-state speakers. Dr. DeCesare seconded the motion and motion passed. Dr. Strinden also moved that the Council authorize expenditures to be allowed the Chairman of the Scientific Program, for telephone and other expenses in setting up a program, and that the Chairman submit the bill to the State Office for Payment. This motion was unanimously agreed upon.

Dr. Dahl advised that the House had recommended that the Secretaries of each District Society be urged to bill collective dues for the Woman's Auxiliary and the Physician's dues for the Association. Dr. Mahowald stated that this is not mandatory, but it is left to the option of the District Society Secretary. The Council reaffirmed their last year's recommendation about billing the collective dues, and it was decided that the Council would send a letter to each Secretary that this is their recommendation to the local Society.

Dr. Dahl stated that the House of Delegates approved the A.M.A. Delegate's request that the North Dakota Medical Association favor the dues increase from $45.00 to $70.00. Dr. Mahowald advised that this matter would be tabled for action by the Council after the final action by the A.M.A. House of Delegates in June.

Dr. Dahl then advised that the House of Delegates requested the Council to create a Foundation for application for a grant for the Feasibility Study on Public Law 89-239; Heart Disease, Cancer and Stroke; and that the majority of these physicians on this committee be practicing physicians. After a lengthy discussion, Dr. Strinden moved that the Executive Committee of the Council and the President of the State Association be authorized to appoint a committee of interested parties to apply for a grant for a Feasibility Study for the program of Heart Disease, Cancer and Stroke; Public Law 89-239. Dr. DeCesare seconded the motion and motion passed.

Dr. Dahl then advised that the House of Delegates had requested the Council to direct the Executive Secretary of the Association to write a letter, signed by the President of the Association, to each of the 50-Year Members who were not here and did not receive their 50-Year pins and Certificates. Mr. Limond advised that this would be done this year, as it has been done in the past.

Dr. Dahl advised the Council that the House voted they were in accord with the subscription of $100.00 to the Science Fair. Dr. Mahowald stated that the Council takes note of this directive from the House and sees no reason for changing any past action and that this will be continued.

Dr. Dahl advised that the House passed a Resolution that the Director of the Crippled Children's Bureau present a list of the procedures covered and their approximate cost be sent to all physicians in the state so

that minor conditions might be eliminated and that acceptance, or non-acceptance of other conditions can be better made at a county level. Dr. Strinden moved that this be carried out as quickly as possible; Dr. Foster seconded, and motion passed.

At the suggestion of Dr. Mahowald, Dr. Strinden moved that the Past President, the Speaker of the House, and the Councillors who are leaving their offices, be presented with a gift from the Association such as a plaque, gavel or some suitable gift, beginning with the present past President, Speaker and Councillors, to be purchased and inscribed at the discretion of the Executive Secretary; Dr. Fox seconded this motion, and motion passed.

Dr. Strinden stated that it had been brought to his attention that on several occasions the Workmen's Compensation Bureau premiums to the Clinics listed all employees the same, regardless of what they are doing, and the premiums to clinics are considerably higher than others for people doing similar work. Dr. Strinden feels that the Medical Economics Committee should instruct Mr. Daner to look into this matter. He then moved that there should be some consultation with the clinic manager and this should then be referred to the Medical Economics Committee. Motion seconded by Dr. Van der Linde and motion passed.

Dr. Strinden feels that there should be an arbitration committee to arbitrate differences between the insurance carrier, particularly of the government programs, and the individual physician, relative to a fee charge. He also feels that the Council should be a grievance committee only in matters pertaining to medical misconduct. Dr. Mahowald informed the Council that Mr. Daner is working on this and would have considerable more information for the Council at the Interim Meeting in December.

Dr. Christianson reminded the Council that there should be another Director for Blue Shield appointed by the Council to replace him. Dr. Mahowald advised that they could appoint one or two Councillors to Blue Shield to replace Dr. Christianson, and if they wished, to replace himself. Dr. Strinden moved that Dr. Fox serve on this Blue Shield Board and Dr. Mahowald continue to serve on the Board. Dr. McLean seconded this motion and motion passed.

Dr. Mahowald said that he felt a letter should be sent to the members leaving the Council; namely, Dr. Naegeli and Dr. Christianson, to the effect that the Council is extremely grateful for their efforts, their wonderful spirit and cooperation while Councillors; along with the plaque to be given to them. This was unanimously agreed to by the Councillors.

There being no further business, the Council adjourned at 6:00 p.m.

INTERIM MEETING

Saturday, December 10, 1966, Fargo, N.D.

The meeting was called to order by the Chairman, Dr. R. E. Mahowald at 10:00 a.m. Roll was called by the Secretary and the following Councillors were present: Doctors R. E. Mahowald, Chairman, R. W. McLean, Keith Foster, W. R. Fox, R. B. Tudor, Dean Strinden, F. A. DeCesare, C. J. Klein, J. M. Van der Linde. The absent Councillor was Dr. R. S. Larson of Velva. Dr. L. A. Giltner of Minot was present as an observer for Dr. Larson.

Others attending the meeting were Doctors: R. D. Nierling, Wm. Buckingham, V. J. Fiseher, C. H. Peters, Dean Harwood, R. C. Painter, T. E. Pederson, P. O. Dahl, Lee Christoferson, Jack Whisnant, G. W. Toomey, and Margaret Fremming and Mr. Sherwood Williams of the A.M.A.

The first order of business on the Agenda was the reading of the minutes of the previous meeting of the Council. Dr. McLean moved and Dr. Strinden seconded, that the reading of the minutes be dispensed with and that they be accepted as printed in the Council book. Motion passed.

Dr. Mahowald asked Dr. R. D. Nierling, Treasurer, for a report of the Association finances to November 30, 1966.

Dr. Nierling discussed the financial status of the North Dakota State Medical Association, as printed on page 14 of the Council Agenda. Dr. Nierling advised also that by following the suggestion of the Council and purchasing C.D.'s of three months duration, the Association received $564.39 in interest. This is a very worthwhile procedure and he would advise to continue to do so, providing he can get the C.D.'s on a three months basis. He asked for some action or discussion on the matter of auditing the books. This past year the books were audited by one accountant in Bismarck and this has proved to be quite inconvenient because the books from Jamestown had to be sent to Bismarck.

There was some discussion by the Councillors of the statement of receipts and disbursements, (page 14—Council Book) and an explanation by Dr. Nierling and Dr. Mahowald of the expenditures of the 1966 budget. Dr. Strinden moved that the Executive Committee of the Council be authorized to work out a most feasible plan for the auditing of the books. Dr. McLean seconded the motion, and motion passed.

Dr. Mahowald then reviewed the proposed budget for 1967, item by item. Dr. Foster made the motion that there be an increase in Mr. Limond's salary of $750.00 per year; this amount to be paid on the premium of Mr. Limond's Retirement Insurance. In other words, the Association to increase the amount they pay towards this premium, by $750.00 per year. Dr. Tudor seconded the motion. Motion passed. Dr. Foster moved, and Dr. DeCesare seconded, that Office Stenographer's salary No. 1 and Office Stenographer's salary No. 2, be increased 5%, and that this be made retroactive to January, 1966. Motion passed. The balance of the items in the proposed budget remain the same with the exception of the Social Security Tax which will be increased by the salary increases, and the premium on the life insurance.

Dr. DeCesare moved to accept the proposed budget as amended. Dr. Tudor seconded the motion, and motion passed. (The approved budget appears at the end of this report).

Dean Harwood addressed the Council and reviewed the actions of the Committee on Heart Disease, Cancer and Stroke, which met on Friday night in Fargo. There was a good representation from around the state at this meeting and tentative plans were discussed as the groundwork for making application for a grant for the Feasibility Study, Law 89-239, Heart Disease, Cancer and Stroke. A representative from the Minnesota Committee appeared at this meeting and advised North Dakota of the program instituted in Minnesota; namely, the Northland Regional Medical Program. Nebraska and South Dakota have a program set up also and have asked North Dakota to join with their program.

Dr. Christoferson then introduced Dr. Jack Whisnant of the Mayo Clinic, Rochester, Minnesota. Dr. Whisnant presented the Minnesota Plan, called the Northland Regional Medical Program. He reviewed the Bill, Law 89-239, which provides two phases of grants; one is a planning grant for regions in any part of the country, and

the other providing an operational grant for these same regions. This is almost entirely a non-directive bill and this part is attractive to physicians. This will develop a whole new era of medical care and the doctors should be a part of this. The physicians are committed to the role of health planning, and that is one of the reasons the State Health Departments have been encouraged to be a part of this plan.

Dr. Whisnant advised that the Minnesota Planning Group is not trying to incorporate any other group that does not wish to be in this, but if North Dakota should so desire to become a part of the Northland Regional Medical Program, it would be necessary for the physicians in North Dakota to do their own planning at a community level. The needs must be described at individual community levels and not directed from some administrative group. The Northland Regional Medical Program was approved for a Planning Grant on November 28, 1966. The next step for Minnesota, therefore, is to hire a Director. Dr. Stickney was appointed Co-ordinator of the program, but he does not intend to be the Director. If North Dakota should desire to be a part of this program, they should also be a part of putting the framework together for the whole region. Dr. Whisnant then explained what the Planning Grant covers. He advised also that Dr. Christoferson and Dr. Harwood had been asked to serve on their Advisory Committee and that they have representatives on their Advisory Group from Western Wisconsin and Northern Iowa. It is important that each region knows what the other regions are doing.

Dr. Christoferson addressed the Council and reported on the Committee meeting held Friday night, December 9th. He asked two questions of Dr. Whisnant; namely, if North Dakota went ahead and submitted the Application for a Feasibility Study Grant, and received this Grant, would it be possible to coordinate the North Dakota Feasibility Study with the Northland Regional Medical Program; and what does Dr. Whisnant think about the aspect of North Dakota providing an individual Feasibility Study. Dr. Whisnant, in answer to these questions, advised that North Dakota could actually put together a Planning Group and apply for a grant, but under the qualifications for receiving this Feasibility Study Grant, it is necessary to have a medical school involved in post-graduate medical training, among other things. It is not likely that a planning grant would be approved for North Dakota as they do not fit in this category. Dr. Whisnant felt also that pressure would be put on North Dakota to integrate with some other program. However, the State must do their own planning for the Feasibility Study Grant, and if North Dakota decided to join the Northland, it might be necessary for them to put together a planning program and make this a supplemental grant for the Northland Regional Medical Program. It may be that there are sufficient funds to do the initial planning necessary.

This plan was discussed at some length and questions were asked of Dr. Whisnant and his advice asked on the procedure. Dr. Christoferson informed the Council that the Committee has a plan whereby they can produce an application, perhaps by February. This plan encompasses Dr. Harwood as a Program Director; and utilizes other successful plans as a guide. This has already been incorporated and a fiscal agent must be chosen to handle the funds, if North Dakota receives the Grant. Dr. Christoferson also asked the Council to make available some funds for completing this study. There will be some expenses, such as long distance telephone calls and some secretarial work. Dr. Harwood and Dr. Christoferson will be in Washington on January 5th when the Regional Planning Group is meeting to discuss an application for North Dakota. When they return from this meeting they will have more information as to the success North Dakota might have to receive a planning grant.

A discussion followed as to money involved in making the study for the application. Dr. Christoferson also advised that the State Health Department has agreed to take an active part in the program and these resources could be used in the study of improving medical resources in the State and to inventory the medical resources.

Recess at 12:45 for lunch.

At 2:00 p.m. the Council reconvened. There was declared a quorum present and Dr. Mahowald called the meeting to order.

Dr. Mahowald asked if the Council should authorize the funding of the luncheon for the House of Delegates on Sunday. It was suggested that this be tabled and no action was taken on this.

Dr. Mahowald brought up the report of the Chairman of the Committee on Medical Economics, as Dr. Lindelow, Chairman, was not present. There was some discussion on the "usual and customary charge." Mr. Sherwood Williams of the AMA defined the "usual and customary charge." A report from the Committee on Crippled Children was also read, as follows:

"The C.C.S. Committee goes on record as approving the philosophy of providing the best medical services possible for children by the continuation of present C.C. Services and extension of aid to the medically indigent within the fiscal limitations of providing an adequate but conservative program."

Dr. Mahowald suggested that this be referred back to the Economics Committee for any action they see fit.

Dr. Fischer informed the Council that some doctors had received payment for services to Welfare patients on the old basis and he felt that a notice should be sent to the doctors and to the Welfare Boards of each county that the Public Welfare Board had agreed to use the "usual, customary and reasonable fees" commencing October 1, 1966, and that they would accept the "reasonable fees" commencing October 1, 1966, and that they would accept the Relative Value Guide to be published in October 1966. Dr. Strinden suggested that these cases aforementioned be documented and sent to the Medical Economics Committee who should in turn go to the Welfare Board and straighten out this matter. Dr. Strinden moved that there be some directive from the State Office to the County Welfare Boards in the immediate future. Dr. Fox sconded the motion and motion passed.

There was an extensive discussion on the ODMC Contract, the Indian Bureau and the Workmen's Compensation fee payments. Dr. Mahowald advised that no action is necessary by the Council, other than the Council supports this action by the Committee on Medical Economics. Dr. Strinden moved that the Council take note of the excellent work done by the Committee on Medical Economics. Motion passed.

Dr. Mahowald then asked Dr. Christoferson to finish his report to the Council of the action taken by the Committee on Heart Disease, Cancer and Stroke. Dr. Christoferson stated that the action taken by the Committee was to recommend before the Council that North Dakota proceed with an application for a grant for a Feasibility Study. He outlined the plan and advised that there would need to be an inventory made in the State

of our resources and how they can be favorably expended in the program of education and care of patients; also how this might fit into a regional type program. It is generally believed that North Dakota would not likely be approved for a Feasibility Study Planning Grant, and that there would be no chance for an Operational Grant and that North Dakota would have to join another program in this area. It was unanimously thought that the Northland group would be the best one to join, as it fitted North Dakota better geographically. It was, therefore, the suggestion that North Dakota proceed with the application for a Feasibility Study Grant and that the Dean of the Medical School be appointed as Program Coordinator, and that the Medical School be appointed as the Fiscal Agent. Dr. Christoferson felt that it would be advisable for the Council to judge whether this is correct, and whether the Committee could proceed on this basis.

It was further felt that the Committee should try to utilize the State Board of Health facilities. However, there would have to be some output of funds by the Association to carry on this study to make this application. The Committee all agreed to work as members of the Committee in this application, if the Council goes along with this. They felt that if the responsibility for a Feasibility Study became that of another regional program, they would sit back and take no part in this, and that it would be hard to get men in our state to study and inventory this. It would be better to go ahead, even though the chances for acceptance in this are not very good. Dr. Christoferson does feel from a personal standpoint that this would be an important thing for the State to do. This Committee would go ahead and work hard to get a reasonable study made; and could use the State Health Department resources to defray the cost somewhat. The doctors' time inventorying this would be donated, but there will be many long distance calls necessary to carry on this program. There is a question now, after talking to the Dean, whether he will have as much time to give this, and it might be well to have an Assistant Program Coordinator such as Dr. Amos of Bismarck, who could devote some of his time and some of his staff and facilities to this. Therefore, Dr. Christoferson, as Chairman of this Committee, requests that the Council support the selection of a Program Coordinator, and a Fiscal Agent; and allot some funds for the development of this application for a Feasibility Grant. Dr. Christoferson pointed out that Dr. Whisnant had stated that the North Dakota bargaining power would be enhanced if they do the survey themselves.

After a great deal of discussion, Dr. Mahowald then brought before the Council the three items the Committee has asked of the Council; namely, 1—a request that the Council support the Committee's choice of a Coordinator; 2—a request for funds in a minimal amount for the use of this Committee; and 3—the approval of the choice of a Fiscal Agent, and a recommendation from the Council to the House of Delegates for consideration.

Dr. Foster moved that the Council support the Committee's choice of a Coordinator; Dr. DeCesare seconded the motion. Motion passed. Dr. Tudor moved that the Council allot funds for the use of the Committee; Dr. DeCesare seconded the motion. Dr. Strinden moved to amend Dr. Tudor's motion to add that the funds be allotted up to an amount of $1,000.00. Dr. Tudor agreed to the amendment and Dr. DeCesare, the second to the motion also agreed to this amendment. Motion passed. Dr. Foster moved that the Council approve the choice of the Medical School as a Fiscal Agent; Dr. DeCesare seconded the motion. Motion passed.

Dr. Mahowald advised that there was no need of a formal vote on the recommendation of the Council to the House of Delegates on this matter.

Dr. Pederson gave a brief summary on the actions of the AMA House of Delegates; namely, asking for doctors to volunteer for Viet Nam; a report from the Chairman of AMPAC that they were successful in 80% of the elections; a report from the Woman's Auxiliary that there is 40% of potential membership to the Woman's Auxiliary and they are asking for help from the physicians of the State Associations to increase this membership; a report from the Student AMA that there were 56 resolutions considered by the House; the matter of the "usual and customary fees" was also discussed, and there was a great deal of discussion on Title 19. The present method of direct billing was strongly supported. The matter of the Utilization Review Committees was taken up by the Legislative Committee and the State Associations are urged to pass state legislation to provide the physicians serving on these committees immunity on any actions taken while serving on these Utilization Review Committees. Regarding Medical Education, it was suggested that the Medical Education Committee of the State Associations review the Committee actions and instruct the delegates prior to the June meeting. The AMA feels that the state associations are going to have to help the nurses, particularly in North Dakota, to obtain a better wage. The AMA Educational Research Foundation is in a bit of trouble because of the tight financial situation. North Dakota does not rate very highly in giving to the AMA Educational Research Foundation. Its record is not too good. TWX is being discontinued as it was too expensive and the little that it was used did not justify the cost of this. Elderly applicants' eligibility for drivers' licensure and drivers' insurance was discussed. It was decided that the physician could be concerned only with whether the applicant meets these requirements; and whether he should be insured or licensed is the responsibility of other agencies. In regard to direct billing to welfare patients, this was brought up and it is to be encouraged wherever possible.

Dr. Buckingham informed the Council that there would not be a great many bills on legislation to bring before the House, as these will not all be in until January 20th. He advised that if the Aid Station at the Legislature is desired, he would start now to take names of those who wish to serve. He also advised that the Legislation Committee is interested in getting a profile on each legislator and he has a form which gives the name, age and pertinent information regarding the legislator. The Committee would like to pass these out and have them filled out in the various areas by those of the physicians who know their legislators. Dr. Buckingham recommended that these be distributed by the Councillors to the physician in each town who is acquainted with the legislator. He also asked the permission of the Council to place in the hands of the legislators, a pamphlet on the Educational Background of Chiropractic School Faculties, before the legislators meet in Bismarck in January, so that they can read and study this. The Chiropractors are trying to put through a bill that they can be called "physician." The doctors are not fighting them, but do not feel that the Chiropractors are qualified to be called "physicians."

Dr. Foster moved to approve the distribution of this pamphlet by the Legislative Committee to the House

of Delegates and to the legislators; Dr. McLean seconded the motion. Motion passed.

A discussion was held on the Abortion Bill that will be introduced at the Legislature. Dr. Mahowald ruled that the Council should take no action on this as the House of Delegates is the voting body.

Dr. Gale Richardson presented a Resolution to the Council as follows:

RESOLUTION

WHEREAS: mechanisms for insuring the quality of medical practice in all its aspects, including clinical pathology, already are adequately provided for under the Medical Practice Act, and implemented by the Board of Medical Examiners, and

WHEREAS, the North Dakota Society of Pathologists reaffirms its support of existing programs under the Joint Commission on Accreditation of Hospitals, and the College of American Pathologists, to upgrade laboratory standards; and that the proposed laboratory licensing law currently being considered by the Legislative Research Committee is a needless duplication of these efforts.

THEREFORE, BE IT RESOLVED, that the North Dakota Society of Pathologists is opposed in principle, to a laboratory licensing act at this time, and will continue in its efforts to insure that all laboratories providing services to patients in this state, meet at least minimal standards.

Signed: Gerard J. Obert, M.D.,
Secretary-Treasurer: North Dakota
Society of Pathologists
December 10, 1966

Dr. Richardson explained the reason for this action and the position taken by the State Society of Pathologists. This resolution was discussed and the bill was interpreted to the Council. It was stated that the Pathologists would like to kill this bill in Committee, and all like bills.

Dr. Richardson presented another resolution passed by the Society of Pathologists as follows:

RESOLUTION

WHEREAS, it is the stated policy of the American Medical Association and its constituent societies, that solicitation is unethical, and:

WHEREAS, it also is a principle of these same societies that competition on the basis of fees is unethical and undesirable,

THEREFORE, BE IT RESOLVED, that the letter dated November 2, 1966, sent by W. E. Cornatzer, Ph.D., M.D. on the letterhead of the University of North Dakota, soliciting Protein-Bound Iodine specimens and offering them at a fee which not only is below prevailing fees in this state, but which is probably below the actual cost of performing the procedure; is against the principles of medical ethics, and,

BE IT FURTHER RESOLVED that the North Dakota Society of Pathologists calls this to the attention of the North Dakota State Medical Association for information and whatever action it deems advisable.

Signed: Gerard J. Obert, M.D.,
Secretary-Treasurer: North Dakota
Society of Pathologists
December 10, 1966

Some discussion was held as to whether this should go directly to the Grievance Committee. Dr. Richardson and Dr. Obert both appeared before the Council to explain this Resolution and the reason for this. Dr. Mahowald asked the Council if they wished to take any action on this matter. Dr. Tudor was of the opinion that if the Council were to consider the matter as a Grievance Committee, that it should be in an executive session and that both parties should appear. As one party of the controversy is not a member of the Association, nor a practicing physician, there was a question as to whether the Council or Grievance Committee had a right to judicate a Ph.D. Dr. Harwood advised that he was certain that this man would conform with the wishes of the Association, as he did not feel that the intent of the letter was solicitation of business, but was only meant to be one of service.

Dr. Mahowald asked Dr. Harwood if he had anything he would like to add to his report on "Inflation, Legislation and the Medical Center". Dr. Harwood explained in detail the reason for this request for an appropriation of funds; particularly as they are not paying the teaching staff of the Medical School the median, and he feels that they should pay at least the median for their instructors.

The next item on the agenda was the appointment of the Board of Directors for COMPACT. Dr. Mahowald advised that Dr. Dahl had agreed to accept the Chairmanship "pro tem" of this Committee until the Committee can appoint their own; the Council does not select the Auxiliary member who is now Mrs. Dahl; Dr. Ulmer has agreed to serve again; Dr. R. S. Larson of Velva has asked that he be removed from that Committee. Dr. Giltner is here and Dr. Mahowald suggested that Dr. Giltner serve on this Committee. Dr. Giltner agreed to serve. Dr. Powers and Dr. Hanewald have agreed to serve. Dr. Christianson is apparently not interested as Dr. Mahowald has received no answer to his communication.

Dr. Klein of Valley City suggested that another letter be written to Dr. Christianson before choosing a member to replace him.

Dr. DeCesare moved that the following slate of Committee members as stated by Dr. Mahowald be elected: Doctors Dahl, Giltner, Hanewald, Ulmer, Elsworth, Powers and Christianson. Doctor Foster seconded the motion. Motion passed.

Dr. Mahowald then asked if it would be satisfactory for the Executive Committee to set up an arrangement for information from the Legislature meetings, and bring its finding to the Council. Dr. DeCesare suggested that this be discussed at a closed meeting of the Council, and asked if Mr. Daner could also meet with the Council. This was decided upon.

Adjournment at 4:30 p.m.

Respectfully submitted,
(Signed) R. E. Mahowald, M.D.,
Chairman of the Council

REPORTS OF COUNCILLORS

First District

The First District Medical Society held eight meetings in the last fiscal year, from March 1966 through February 1967. These meetings were all held at the Oak Manor Motor Lodge in Fargo. The March meeting was held on March 28, 1966 and was called to order by the President, Dr. Blaine Amidon.

Following the format adopted last year, the scientific portion of the program was put on first, and this was followed by the business of the Society. Dr. Lyle J. Hay, clinical professor of surgery at the University of Minnesota, gave a very interesting paper entitled, "Selection of Operation for Peptic Ulcer Patients". A very lively discussion followed the presentation of this paper.

The business meeting was then conducted and the application of Dr. Allan J. Schutt was read for the first time. The application of Dr. Robert L. Leegard was read for the second time. The applications of Dr. Ross Halliday and Dr. Ann Schutt were read for the third time, and on written ballot, Drs. Halliday and Schutt were unanimously elected to membership.

In the line of communications, the secretary read a letter from Dr. John Gillam, announcing the formation of the new Fargo Medical Foundation with its main offices in Fargo, North Dakota. A letter was submitted to the secretary. In asking for committee reports, Dr. Arthur Burt reported for the Economics Committee and Dr. Lamb reported for the Public Relations Committee. It was moved by Dr. Seifert and seconded by Dr. Donat

that we change the location of our meeting places as of September 1966. The motion was amended to read that the change will be made after December 1966. After much discussion, it was suggested by Dr. Seifert that we use sites in both Moorhead and Fargo, and the consensus of opinion was in agreement. The amendment and the motion passed. Dr. Amidon reported briefly on a meeting held in Jamestown March 26, 1966 with regard to the proposed Heart Disease, Stroke and Cancer Government program. He stated that a Committee was to be appointed to study the feasibility of such a center in our state. He also reminded the membership to be prepared to advise delegates at the next meeting, and that Committee reports will be called for. The meeting was adjourned at 9:10 p.m.

The next meeting of the First District Society was held on April 18, 1966 at the Oak Manor Motor Lodge in Fargo. The meeting was called to order by the President, Dr. Amidon. At this meeting the following resolutions were passed as instructions for the Delegates to the House at the State Meeting. One resolution passed was to liberalize the present abortion law in the State. A second resolution was that the number of delegates to the House from each District be proportioned to the paid membership of the District as of March 1st of the current year. The third resolution was for a committee to study the feasibility of a center for treatment of Heart Disease, Stroke and Cancer, and that this Committee have a majority of practicing physicians. The last resolution was that money should be provided for honorarium to speakers at the State Convention who are not members of the North Dakota Medical Society. There being no further business, the meeting adjourned.

The First District Medical Society does not hold a regular meeting in the month of May as this is the month in which the State Medical Association meets. Consequently, the next regular meeting of the First District Medical Society was held on September 26, 1966. This meeting was preceded by a meeting of the Executive Committee consisting of Drs. Amidon, C. M. Hunter, L. G. Pray, J. Schneider and Henry Norum. At this committee meeting, the following items were discussed, including the proposed change of the abortion law, membership dues, choice of meeting places for 1967 and community health week. Following this, the regular meeting of the District was called to order by the President, Dr. Blaine Amidon, at 7:30 p.m. The minutes of the preceding meeting were not read in the interest of time. The applications of Dr. Leland Larson and Michael Barnes were read for the first time. The applications of Dr. John Rawland, Dr. Jack Glover, Dr. Jane Magill and Dr. Charles Stevens were read for the second time. The application of Dr. Allan Schutt was read for the third time. A written ballot was taken, and Dr. Allan Schutt was unanimously elected to membership.

The scientific portion of this meeting consisted of a very interesting paper on anemia. This paper was presented by Dr. Mathew Block, Professor of Medicine, University of Colorado Medical School.

As is the custom at this September meeting, the President of the North Dakota State Medical Association, Dr. Glenn Toomey, spoke to the members of the Society. He discussed many timely topics as they applied to the practice of medicine. He strongly recommended our cooperation during the legislative session next January and also urged our membership in AMPAC and COMPACT.

Mr. Lyle Limond reported on a recent agreement made by the State Board of Welfare in the letter to the State Medical Economics Committee. They have agreed to pay the usual and customary fees starting October 1st, 1966, running until June 30, 1967. The old rate will apply from July 1st to October 1st, 1966. He further stated that on the Crippled Children's Services, a lower rate schedule will remain in effect. Dr. W. E. G. Lancaster spoke of the need to appoint a physician of this Society to meet with the Cass County Mental Health Board. He, himself, had served in this capacity for some 30 odd years and because of his retirement, a successor must be named. Dr. Kohlmeyer expressed an opinion that it should not be a psychiatrist so that there would not be a conflict of interest. The President offered to pursue this matter further. Dr. Donat informed the society of a series of programs which would be presented by the Educational TV station for the physicians in this area. They would be shown from September through May 1967. Dr. O'Hearn recommended that the Society endorse the Junior League program of checking pre-school hearing and vision. A motion was made and seconded and passed so that the Society would endorse the program. Dr. Christu spoke before the group and discussed plans for Community Health Week beginning October 16th. Public Health Committee proposed a campaign to educate the public in regard to poisonings and the uses of syrup ipecac.

The Secretary read a communication from the President of the Woman's Auxiliary stating that they were willing to help the Society in its membership in any way possible. The proposed changes in the abortion law were discussed. Certain recommendations had been made by the Maternal and Child Welfare Committee. This discussion was tabled until the October meeting. Dr. Ulmer also spoke for the support of AMPAC and COMPACT. The meeting adjourned at 10:00 p.m.

The next meeting was called to order by the President, Dr. Blaine Amidon, at 7:30 p.m., October 31, 1966. The minutes of the previous meeting were read and approved. Application of Dr. R. B. Harrington was read for the first time and applications of Dr. Leland Larson and Dr. Michael Barnes were read for the second time. The applications of Dr. John Rawland, Dr. Jack Glover, Dr. Jane Magill and Dr. Charles Stevens were read for the third time. A written ballot was taken and Drs. Rawland, Glover, Magill and Stevens were unanimously elected to membership.

Dr. Perry Engstrom, a member of our Society, spoke on his experiences in Viet Nam. The talk was illustrated with colored slides and was most opportune, interesting and well received. Dr. Engstrom encouraged any and all physicians interested, to consider such an experience. The project under which he went to the Far East is known as Volunteer Physicians for Viet Nam.

Discussion at this meeting again was held concerning proposed changes in the abortion law. It was moved, seconded and passed that the changes of the proposed law as recommended by the committee on Maternal and Child Welfare be accepted and recommended by our Society. A request came up before the Society to donate a sum of money for the educational television station in Fargo. It was suggested by one of the members that there was a possibility for this television station to obtain educational funds from the North Dakota Division of the American Cancer Society, the North Dakota Chapter of the American Heart Association and perhaps from the Fargo and Dakota Medical Foundations. After considerable discussion this motion for donation of funds was voted on to be tabled and so passed. No further action was taken. It was also decided at this meeting that the December meeting would be changed from December 26th to December 19th. The Secretary then read

two letters requesting endorsement of the First District Medical Society. One letter was from the St. John's Hospital extended care nursing center; the other from the American Lutheran Homes. It was passed by the District Society to endorse both of these projects. Dr. Ulmer invited the members of the Society to attend the Annual Darrow-Long Lecture which will be held November 19, 1966. There being no further business, the meeting adjourned at 10:00 p.m.

The next meeting was held on November 28, 1966, and was called to order by the President, Dr. Blaine Amidon, at 7:55 p.m. Dr. F. A. DeCesare, Councillor the First District Medical Society, presented a plaque from the American Medical Association to Dr. Perry Engstrom in recognition of his voluntary service for Project Viet Nam.

The Scientific Program consisted of a very fine paper presented by Dr. Alex Schirger, a consultant in the section of Vascular Diseases of the Mayo Clinic. He spoke on the "Advances and the Treatment of Hypertension." His paper precipitated a very lively discussion and was enjoyed by all. Following the scientific portion of the program, the minutes of the previous meeting were read and approved. The minutes of the Executive Committee Meeting of November 28th were read and brought up as items of new business. Next on the program, the application of Dr. R. B. Harrington was read for the second time and the applications of Dr. Leland Larson and Dr. Michael Barnes were read for the third time. A written ballot was taken and Drs. Larson and Barnes were unanimously elected to membership.

The next order of business was the consideration given to the amount of money held by the Treasurer of the First District Medical Society. Many demands have been made upon this fund of money, and it was decided by the membership that the First District Medical Society would establish a trust fund. The interest of this trust fund would be donated as prizes and scholarships to worthy medical students at the University of North Dakota Medical School. This motion was amended and three of the members of this Society will act as a Committee and meet with the Dean of the Medical School to implement the plan. The amendment was also passed.

At this meeting it was moved, seconded and passed that the doctors of Clay County, our neighboring county across the river in Minnesota, be invited to attend our meetings and that a letter be sent to Dr. John Holten, informing him of this fact. This action was brought about because of the fact that the Clay County Medical Society which has, in the past, met with the Becker County Medical Society of Detroit Lakes was not working out satisfactorily because of the distance—and their own society, being so small, showed a desire to meet with the larger district; namely, the First District of Fargo.

Next on the Agenda was a considerably long discussion in regard to the Society's wishes as far as the administration of Title XIX funds. Dr. Burt stated that it was his impression that the North Dakota State Welfare Department would like to administer the Title XIX funds. The Legislative Committee of the State Medical Association had not met and the House of Delegate's Interim Session for December 1966 has been cancelled. It was also announced that the House would meet February 5, 1967, which is after the deadline for introducing new legislation in the State Legislature. Dr. Christoferson stated that it was his feeling that the Interim Session could not legally be called off. During this discussion, it was suggested that the Blue Shield and Blue Cross plans be asked to service these Title XIX funds and Dr. DeCesare suggested that the Executive Directors of the Blue plan could be asked to study such a proposal. Dr. Christoferson reiterated again that it was imperative that the House of Delegates meet prior to the introduction of bills into the State Legislature, and that he felt a quorum could be obtained. Final action was taken and it was moved, seconded and passed that the Councillor, Dr. DeCesare, personally call Dr. Glenn Toomey, the State President, Mr. Eagles of Blue Shield and Mr. Laybourn of Blue Cross to see that the proper procedures be followed in regard to Title XIX and meetings of the Legislative Committee and the House of Delegates. Further discussion on the disbursement of Title XIX funds was brought about and Dr. Eric Barth reviewed the experience in New York State. He stated that the medical fees were arbitrarily set at approximately ⅔rds of the Workmen's Compensation fee and some cases were made retroactive. The meeting adjourned at 9:45 p.m.

The next meeting of the First District Medical Society was held on December 19, 1966 and was called to order by the President, Dr. Blaine Amidon. The applications of Dr. Jack Leigh and Dr. Stanley Thompson were read for the first time. The application of Dr. R. B. Harrington was read for the third time. A written ballot was taken, and Dr. Harrington was unanimously elected to membership. Following this, the minutes of the previous meeting were not read after motion duly made—seconded to this effect.

A letter from the Americana Nursing Home stating that they would admit patients without regard to race, color or national origin was noted. Dr. Amidon, the President, reported on a request he had received from Station KFME for a debate between local chiropractors and members of our Medical Society. After discussion, it was decided that the First District Medical Society would not accept this challenge and would contact station KFME and explain to them the reasons. Further business consisted of Dr. Rawland moving that our Legislative Committee meet with the Legislators to introduce a plan to establish a uniform basic science law in North Dakota. This was seconded and passed. Dr. Ulmer reported on COMPACT and stated that the 90th Congress of the United States would have enough moderates and conservatives to form a majority. He called to the attention of the members that AMPAC had been active in the campaign with 146 candidates. (131 for House seats and 15 for Senate seats.) He also briefly reviewed the action of the House of Delegates. Following these discussions, Dr. Amidon called for a report of the Nominating Committee which consisted of Drs. Hugh Thorfinnson, Chairman; Neil Williams and George Thompson. The slate presented by the Nominating Committee was for President, Dr. W. O. Webster; Vice-President, Dr. C. Fercho; Secretary-Treasurer, Dr. H. A. Norum; Board of Censors, Dr. Schneider—three year term to be replacing Dr. C. M. Hunter; House of Delegates, Drs. H. A. Norum, M. H. Poindexter, A. C. Burt, R. E. Kulland, Paul Beithon, B. F. Amidon, R. J. Ulmer and David Jaehning; Alternates, Drs. Gillam, Goltz, Gaebe, Fercho, Wasemiller, Houghton, Williams and Engstrom. It was moved, seconded and passed that the nominations be closed and that a unanimous ballot be cast for the slate as presented by the Nominating Committee. Just before closing the meeting, it was announced that the Executive Committee had studied other possible meeting sites and had decided to continue meeting in 1967 at the Oak Manor Motor Lodge. Dr. Gillam made a motion, which was seconded and passed, that a vote of thanks be extended to Dr. Blaine Amidon for a

successful year as President of the Society. The meeting adjourned at 9:30 p.m.

The next regular meeting of the First District Medical Society was held at the Oak Manor Motor Lodge on January 30, 1967 and was called to order by the new President, Dr. William Webster at 7:50 p.m.

The scientific program consisted of a presentation of Dr. Alvin Schultz, professor of medicine at the University of Minnesota and Chief of Medicine at the Hennepin County General Hospital. He spoke on the "Metabolic Aspects of Obesity" and his talk was very well received and he pursued it further and outlined his experiences with starvation effect in obese patients and the results of bypassed surgery. Following the scientific program the minutes of the previous meeting were read and approved. The application of Dr. Ghatan was read for the first time. The applications of Dr. Stanley Thompson and Dr. John Leigh were read for the second time. In the line of communications, the secretary read a letter from the North Dakota State Department of Health . . . stating that the Chemo prophylactic drugs for patients with tuberculosis were available upon request from the physician. Dr. Robert Ulmer then reported for the Legislative Committee of the State Medical Association. He outlined the status of bills, both in the House and the Senate which were of medical interest. The meeting adjourned at 9:25 p.m.

The last meeting of the fiscal year was held on February 27, 1967, at the usual meeting place and was called to order by the President, Dr. Webster, at 8:00 p.m. The scientific portion of the meeting consisted of a paper presented by Dr. Edward Liten, a psychiatrist and head of the second section of psychiatry of the Mayo Clinic. The topic he chose was "Depression". His paper was very well received and there was a lively discussion following his presentation. Following the scientific portion of the meeting, the minutes of the previous meeting were read and approved. The application of Dr. Said Ghatan was read for the second time. The applications of Dr. Stanley Thompson and Dr. John Leigh were read for the third time. A written ballot was taken and Drs. Thompson and Leigh were unanimously elected to membership.

A motion was presented to the meeting by Dr. Paul Beithon that a resolution be drafted, stating our position on Daylight Saving Time. The motion was seconded and passed that we would send such a letter to the appropriate legislators stating that we were in favor of daylight saving time. An addendum inasmuch as the House had already acted on this bill, a telegram was sent to Governor Guy stating that members of the First District Medical Society were in favor of his veto on this bill.

On the new business, a request was presented to spend up to one hundred dollars as a contribution to the YMCA for sending needy children to camp. Dr. Gillam noted that this type of action should be handled by the Executive Committee, particularly along lines of policy. Dr. Paul Beithon pointed out at this point that doctors outside of the Fargo Area, members of the Society, would undoubtedly be opposed to aiding a local project. He had no quarrel against the YMCA Camp need, and finally after further discussion, this subject was tabled. On the new business, the following item was taken up:

Dr. Ulmer made a motion that a letter be sent to every retail pharmacy asking them not to call physicians for refills after hours except in acute cases of emergencies. This motion was discussed but was defeated. Dr. Paul Beithon discussed a topic which he termed "Phonemanship" and made several good suggestions as to how to expedite long distance calls. The meeting adjourned at 9:15 p.m.

Respectfully submitted,
(Signed) F. A. DeCesare, M.D.,
Councillor, First District

Second District

A total of 8 meetings were held during the fiscal year from March 1966 through February 1967. Due to inclement weather, all meetings were not fully attended. However, excellent attendance was obtained unless weather conditions prohibited. The present staff of officers was elected in January 1967 and are listed as follows:

President	
DR. R. S. L. HILLMAN	Rugby
Vice-President	
DR. E. V. GILBERTSON	Devils Lake
Secretary-Treasurer	
DR. L. F. PINE	Devils Lake
Delegates	
DR. G. H. HILTS	Cando
DR. R. D. McBANE	Devils Lake
DR. C. R. MILLER	Bottineau
Alternate Delegates	
DR. J. W. LIND	Bottineau
DR. S. J. COOK	Rolette
DR. A. NEUENSCHWANDER	Rolla
Censors	
DR. D. G. McINTYRE	Rugby
DR. J. TERLECKI	Minnewaukan
DR. JERRY D. WOLF	Bottineau

The Delegates of the State Association were nominated as Corporation Members to the North Dakota Physicians Service and Dr. K. W. Kihle of Bottineau was re-elected as a Board member to the North Dakota Physicians Service.

March 10, 1966: The quorum was not present at this meeting because of inclement weather. No business was conducted but recommendations were made:

1. Dr. Miller of Bottineau felt that some form of a utilization review committee should be established on the District Society level and thus a more efficient review could be done in the various hospitals where no competitive factor was involved.

2. A letter was read from the Science Fair. The secretary was to write to them expressing the feeling of the Society, that since we come from so many counties, the physicians be contacted in their own areas for contributions.

Communications to Dr. Rinn, President, and Dr. Robert Fawcett from Dr. Harwood in regards to the Heart, Cancer and Stroke Program were discussed. Dr. Toomey and Dr. Fox were appointed to represent our District Society at the next meeting. The program for the evening was sponsored by the Cancer Society and Dr. Theodore Grage, University of Minnesota, was the speaker. His topic was "Cancer of The Extrahepatic Biliary Tree."

April 7, 1966: Meeting was held at Andrew's Steak House, Rugby, N.D. Speaker for the evening was Dr. Burton Olson of the McCannel Clinic, Minot, North Dakota, and his topic was "Office Ophthalmology."

Dr. C. R. Miller discussed his proposal about a District Utilization Committee. The consensus was that we were not too sure just what type of utilization committees are going to be necessary, and it was decided to table this discussion for a future time.

The application of Dr. Morrison was approved by the Board of Censors and passed. It was noted that he had a temporary license.

The Secretary read a letter from Dr. McBane regarding the Cancer, Heart and Stroke meeting and Dr. Fox gave a verbal report regarding the meeting. It was the feeling that some money was to be obtained to make a survey as to what the State requirements would be and it was requested by our Delegates that we approve of this fund at the State meeting.

Dr. Toomey discussed the International Music Camp at the International Peace Gardens and felt that we should underwrite some students at this camp. Dr. Miller discussed the fact that we should be interested in the Science Fair, and a motion was made by Dr. Pine and seconded by Dr. Toomey that a committee be appointed to study the Music Camp and Science Fair angle and give a report at the next meeting.

May 5, 1966: Met at Andrew's Steak House, Rugby, N.D. Vice-President, Dr. Hillman, introduced the program for the evening—Mr. Eagles of N.D. Blue Shield discussed the pertinent information regarding Medicare insofar as such material was available at the present time. Following, Dr. Hillman recognized Dr. Toomey as our new president of the North Dakota State Medical Association. Dr. Morrison, a new physician from Towner, was introduced. The medical students terminating their second year in serving their clerkships in our regional hospitals were introduced. Dr. Hillman brought up the fact that Dr. Grage had requested a $150.00 honorarium, which the Cancer Society at the present time did not wish to underwrite. Motion made by Dr. Toomey and seconded by Dr. Johnson that the secretary write to Dr. C. M. Lund at Williston regarding this matter. Committee reporting on the International Music Camp was made. The Committee had met with Dr. Utgaard, director of the Music Camp, and it was determined that at the present time funds are needed for equipment for a ceramic lab., and Dr. C. G. Johnson made a motion, seconded by Dr. Mahoney, that we send the administration of the Music Camp $200. Motion was made that the meetings of the Society be held in Devils Lake during the months of September, October, November, December and May and that January, February, March and April be held in Rugby. This motion was passed. Following this a report was made by the Delegates, Doctors Hilts, McBane and Miller. Dr. McBane elucidated the points of major interest, namely the abortion laws, the physician, M.D., and Dr. of Osteopathy relationship and pending legislation regarding this and the method of licensing drivers. Dr. Fox gave a report on the Council in regard to the financial situation, etc. Dr. Mahoney suggested that the doctor of the day get more publicity when they go to Bismarck and proper recognition for the services they are rendering. No further business and the meeting was adjourned.

September 8, 1966: Meeting held at the Ranch, Devils Lake, N.D. Our president, Dr. Rinn, introduced the speaker for the evening, Mr. James W. Rosevear, Field Representative for AMPAC from Minneapolis. An interesting report was given on the activities of AMPAC which caused a fair amount of intense discussion.

New business was brought up. Dr. Miller made a motion, seconded by Dr. Mattson that the May meeting be held in Bottineau, namely at the Lake Metigoshe resort and the motion passed. Dr. Rinn presented for discussion the following topics:

1. Utilization Review Committees and the charges for providing our services in implementing and serving on these committees.
2. The welfare situation at the present time, billing for our usual and customary fees and whether our payments have been received, etc.
3. Nursing Home care charges and utilization review committees for these.

No definite commitments were made on the topics discussed.

Dr. Hilts then brought up the problems of the Abortion Law since he and Dr. Mahoney will be attending the OB-GYN meeting in the near future. Dr. Miller, after serious consideration, made a motion that these physicians attending from our Society use their considered judgment in regards to this particular problem and evaluation of the numerous methods available for saving the life of a mother and child under extreme circumstances. Being no further business, the meeting adjourned.

October 6, 1966: Meeting held at the Ranch, Devils Lake, N.D. Meeting called to order by Dr. Rinn. He introduced the program speaker for the evening, Dr. Richard Olafson, Neurosurgeon, who gave an excellent presentation on Head Injuries. Following this, dinner was held and the business meeting was held. In regards to old business, Dr. Mahoney gave a rather concise report on the OB-GYN meeting in regard to the Abortion Law and a comment from Dr. Terlecki was added to this. It ended up that there was nothing concise to present as the committee who were supposed to have done work at this particular phase had not really done much of anything at all. Dr. Pine then reported on the Blue Shield meeting regarding remuneration for anesthesia and assistants at major surgery. Dr. Hillman made a motion that was seconded by Dr. Mahoney that our Society go on record as favoring direct payment to the assistants in general surgery.

An application from Dr. Guy Montgomery which had been approved by the censors was read and a motion made by Dr. Gilbertson and seconded by Dr. Mahoney that it be approved and passed. No further business and the meeting was adjourned.

On November 3, 1966, the meeting was held at the Ranch, Devils Lake, N.D. The meeting was called to order by President, Dr. Rinn. Dr. Stanley Thompson, Radiologist from Fargo, gave an excellent presentation on the various methods of excretory urography. Following this, dinner was held and the minutes of the previous meeting were read and approved. A letter from the State Secretary was read regarding our dues which for the State Association remained at $100. The AMA dues are now $70. A motion was also made that we have three delegates at our next legislative meeting. Dr. Robert Fawcett read a letter from Dr. Haunz regarding the diabetic detection drive. Dr. Miller made a motion that we have a drive; however, because of lack of a second the motion died. Dr. Hillman stated that Dr. Donald Barcome, Director of the N.D. Rehabilitation Center, Grand Forks, will be here to discuss the facilities of the center at our next meeting. He also stated that Dr. Buckingham will present his work as a medical missionary in the Honduras. Dr. Miller gave a lengthy discussion on the measles clinics. However, nothing further was done. There being no further business the meeting was adjourned.

December 1, 1966: Meeting held at the Ranch, Devils Lake, N.D. The meeting was called to order by Vice-President, Dr. Hillman. Speaker for the evening was introduced—Dr. Donald Barcome, Director of the N.D. State Rehabilitation Center, who gave an excellent presentation on rehabilitation. Following this dinner was held and the minutes of the previous meeting were read and approved. Dr. Miller made a motion which was seconded by Dr. McBane that the Devils Lake District Medical Society go on record as supporting a mass im-

munization for measles prior to the end of January. This was passed. The application of Dr. Neville Jones, which had been approved by the Board of Censors, was read. Motion was made by Dr. Mahoney and seconded by Dr. McBane that he be approved and this was passed. Dr. Miller made a motion that the Auxiliary dues be paid by the members of the Devils Lake District Medical Society. This was seconded by both Dr. Toomey and Dr. Mahoney; however, the motion was defeated. A letter from Dr. Marlin Johnson in regard to the programs of the State Medical meeting was read, and he is looking for assistance in developing his program. Dr. Hilts had a few words to say being a delegate, and brought up points from the Academy Meeting of General Practice. There was some confusion regarding Heart, Cancer, Stroke versus Cardio-pulmonary grant. Dr. Barcome, who fortunately was present, stated that the Cardio-pulmonary grant applied for had nothing to do with the Heart, Cancer, Stroke setup. His idea was to take in only patients with established cardio-pulmonary diseases to determine to what extent he had to be rehabilitated and then return them to their personal physicians for total management. He then discussed the fact that the Public Health Center at the University of N.D. had been moved from Grand Forks to Bismarck. Dr. McBane then discussed the possibility of an Abortion Law coming up in the House of Delegates, and a motion was made by Dr. Pine and seconded by Dr. Mahoney, that our delegates use a judicious approach in regards to the Abortion Law at the House of Delegates meeting. Motion was made by Dr. Pine and seconded by Dr. Mahoney that our delegates develop a format following their meeting and this to be distributed immediately to each member of our Society so they can contact the legislators before they go to Bismarck for the coming biannual legislative meeting.

February 2, 1967: Held at Andrew's Steak House, Rugby, N.D. Speaker for the evening was Dr. George Johnson who discussed Alternate Corticoid Steroid treatment and also briefly discussed immunizations. Following this, the dinner was held and the minutes of the last meeting were read and approved. Treasurer gave the annual report stating that the Gate City Savings and Loan Association had $834.90 and that we had a checking account of $374.10. Following this, we had the annual address of the President of the North Dakota State Medical Association Dr. G. W. Toomey.

Thence, election of officers was held. Dr. Hillman was nominated and elected as President. Dr. Gilbertson, Vice-President, and Dr. Pine, Secretary and Treasurer, and the delegates were re-elected. New director on the Blue Shield Board of Directors then came up and Dr. Kihle was re-elected. North Dakota Blue Shield Corporation members were re-elected to represent our group. Dr. McIntyre discussed the new Adolescent Treatment Center now under extensive study in the legislature in the hope that a separate establishment be set up completely separate from our present Jamestown Mental Institution. A motion was made and seconded and passed that Senate Bill 118 be supported that would establish a completely separate Adolescent Treatment Center. No further business—the meeting was adjourned.

Respectfully submitted,
(Signed) W. R. Fox, M.D., *Councillor*
Second District

Third District

The regular monthly meeting of the Third District Medical Society was held at the Bronze Boot Steak House on May 19, 1966. The meeting was called to order by Dr. Robert C. Painter, President, at 8:50 p.m. The minutes were approved as read.

The following committees gave a negative report: Program Committee, Dr. Nelson; Awards Committee, Dr. Tarpley; Medical Review Committee, Dr. Painter; Legislative Committee, Dr. Clayburgh.

The Mental Health Retardation representative, Dr. Helenbolt, made the following requests:

1) The Board of Directors of the Mental Health Retardation Center requests one physician to be appointed to the Medical Records Committee of December. Dr. Painter appointed Dr. George A. Wilson to this post.

2) Dr. Helenbolt recommended that the following letter be sent by the Third District Medical Society to Leslie O. Ovre, Executive Director of the Public Welfare Board of North Dakota, Bismarck, North Dakota. Clarence O. Ohlsen, County Director, Grand Forks County Welfare Board, Court House, Grand Forks, North Dakota, and O. Victor Lindelow, M.D., Chairman of the Economics Committee, Bismarck, North Dakota.

Being aware of the complete disregard for psychiatric compensation on a Welfare basis, despite if he is scheduled for psychiatric hospitalization and realizing that the State psychiatric facilities are available at Jamestown (State Hospital) and at Bismarck (State Clinic for Children), we hereby go on record as recommending adoption of a psychiatric fee schedule for qualified private practitioners of said specialty. In accordance with their recognized local effectiveness in at least the emergent and/or short term diagnostic situation.

It was moved by Dr. Evans and seconded by Dr. Sandmeyer that this letter be sent as read, and the motion was passed.

Dr. McLeod then made a report on the Rehabilitation Center Committee to the effect that it was desirable as a certification of the Hospital, as a Hospital, that the Rehabilitation Center have an organized medical staff. This was moved that the doctors of the Third District support this activity in the establishment of a regular medical staff and a report of this action be sent to Dr. Theodore Harwood, and to Dr. P. L. Owens.

Report of the Delegates to the North Dakota State Medical Association was then given and reviewed. A report by the Councillor, Dr. Mahowald, was given and reviewed.

New Business: Operation Head-Start was explained by Dr. Tarpley, and the required physical examination situation gone over, and a final move by Dr. Evans and seconded by Dr. Silverman that the Society be willing to do the examinations at the usual customary fee. The motion passed.

After several other points of discussion, the meeting was closed and the President accepted the motion of adjournment.

The regular meeting of the Third District Medical Society was held in the Eagles Club Room of Grafton, North Dakota, on September 15, 1966. This meeting was called to order by Dr. R. C. Painter, President, at 8:35 p.m.

The program was presented by Dr. John Farr, Winnipeg, Manitoba, on acute and chronic arterial insufficiency of the leg. It was very well received and extremely well delivered.

The only new business which came up for discussion was that of the membership of Dr. Robert C. Warner, which was unanimously passed and the ballot was unanimous that he become a member.

There being no further business, the meeting was adjourned.

The regular meeting of the Third District Medical Society was held on October 20, 1966. The meeting was called to order by the President-Elect, Dr. Harold I. Tarpley at 9:00 p.m.

The program was then presented by Dr. Lucien A. Smith on the topic "Pain Patterns and Various Abdominal Conditions." The Business meeting then followed.

Motion was made and passed to dispense with the reading of the minutes. On a motion of Dr. Powers, and seconded by Dr. Nelson, rules were suspended and a unanimous ballot was cast in favor of accepting the application of Dr. Edward M. Johnson of Cavalier, North Dakota into active membership.

Motion was made by Dr. Powers and seconded by Dr. Helm that a diabetic detection drive be instituted this year as outlined in the November, 1965 meeting. Dr. Robert C. Warner was appointed Chairman by the Chair and a contribution of $200 by the Society towards this drive was passed. A letter was read from Mr. W. R. Hanson of the Grand Forks Association of Life Underwriters concerning the MEDIC ALERT program. It was moved by Dr. Powers and seconded by Dr. Evans that this Society endorse this program and the President to appoint a committee as recommended by Mr. Hanson. The motion was passed.

A letter was then read from Dr. Barcome concerning the "Cardiopulmonary Unit" at the Rehabilitation Center, requesting the support of the Society. This letter was read by Dr. Evans. A subsequent motion by Dr. Wilson, seconded by Dr. McLeod that this Society give wholehearted support was endorsed and the motion passed.

Dr. McLaughlin asked support of the Society for a coming amendment to be voted on concerning the tax levy for the Mental Health and Retardation Center. It was moved by Dr. Haunz, seconded by Dr. Powers, that we give our unreserved support to this amendment. This motion was passed.

The meeting was adjourned at 10:15 p.m. on the motion of adjournment.

The regular meeting of the Third District Medical Society was held on November 17, 1966.

The reading of the minutes was dispensed with on a motion by Dr. Powers and seconded by Dr. Leigh and carried.

The new Relative Value Guide was introduced. The Delegates to the North Dakota State Medical Association were instructed to give their support to this new guide as were the members of the Blue Shield Corporation.

It was moved by Dr. Powers, seconded by Dr. Wallace W. Nelson, that the rules be suspended and a unanimous ballot be cast in favor of accepting the application of Dr. William Goodall of Langdon, North Dakota. This motion was passed.

At the invitation of the Society, Mr. Arthur Gustafson of the Public Health Laboratory in Grand Forks was then asked to discuss some of the recent changes that have been suggested in the Public Health Laboratories of the State. Mr. Gustafson pointed out that there was a strong move on certain people's part to move the entire laboratory to Bismarck. It was the general feeling of everyone present that this would be detrimental to the eastern half of the State, The Medical School and the University, and was probably unnecessary. It was moved by Dr. Powers and seconded by Dr. DeLano that the President be instructed to send a letter to the State Health Officer, our legislators, members of the State Health Council and the LRC indicating our support for the maintenance of the Public Health Laboratory at its present location. It was moved by Dr. Keith Vandergon and seconded by Dr. Helm that the President appoint a committee to follow up on this and that the committee be empowered to take whatever action seemed necessary.

Mr. Oscar E. Manz, Director of the Northeast District Science Fair, requested our support of the local Science Fair. It was moved by Dr. Woutat, seconded by Dr. Vandergon and Evans, that the Society support the Science Fair with a $100.00 donation. This motion was passed.

The regular meeting of the Third District Medical Society was held at the Bronze Boot Steak House, Grand Forks, on December 15. The meeting was called to order by the President, Dr. R. C. Painter at 8:35 p.m.

Dr. Lee A. Christoferson then gave the scientific program which was a paper on the "New Surgical Approach to the Disc Problem." This was followed by discussion and questions.

The Committee appointed by Dr. Painter for the Public Health Laboratory problem consisted of Dr. Lambie, chairman; members Drs. Vandergon, Powers, Eelkema, and Clayburgh.

Reports were then given by the Delegates and the Councillor of the Interim House of Delegates Meeting and the Council meeting at Fargo. A number of points were discussed, but no action was taken, as none deemed necessary at this time.

It was announced that a memorial was sent to the Mental Health and Retardation Center in the name of Dr. H. Angus Bowes, a recently deceased member.

Dr. Barcome then gave a committee report on the Mental Retardation Committee. He did not indicate how many meetings this entailed, but did state that (1) the University of North Dakota had applied for a grant and (2) that there was an approved grant for the Mental Retardation Center at the Rehabilitation Center primarily for evaluation and diagnosis.

The President then appointed the Nominating Committee consisting of Dr. Sandmeyer, chairman; Drs. Tarpley, W. W. Nelson, and R. E. Mahowald as members of that Committee. The Auditing Committee consisted of Dr. Helenbolt, chairman; and Dr. Evans.

The meeting was adjourned.

The regular meeting of the Third District Medical Society was held January 19th at Grand Forks, North Dakota. The meeting was called to order by Dr. R. C. Painter, President. Dr. Kenneth F. Swaiman, University of Minnesota Department of Pediatric Neurology, presented an excellent paper on "Seizures in Childhood." This was followed by discussion and questions, and was well participated in by the group.

North Dakota State Medical President, Dr. Glenn W. Toomey, was then introduced by Dr. Painter, who gave several brief comments on problems that faced medicine as he saw them.

On a motion by Dr. Silverman, seconded by Dr. Clayburgh, the minutes were accepted with reading. Motion carried.

The report of the Nominating Committee was as follows:

President, Harold I. Tarpley, M.D.; President-Elect, Wm. C. Nelson, M.D.; Vice-President, N. J. Kaluzniak, M.D.; Secretary-Treasurer, Thomas L. Schafer, M.D.

Delegates to the North Dakota State Medical Association:

John A. Sandmeyer, M.D.; George L. Countryman, M.D.; Richard H. Leigh, M.D.; Kenneth S. Helenbolt, M.D.; Harold W. Evans, M.D.; William T. Powers, M.D.

Alternate Delegates to the North Dakota State Medical Association:

Wallace W. Nelson, M.D.; William P. Teevens, M.D.; B. J. Clayburgh, M.D.; Thomas L. Schafer, M.D.; Norman M. Helgason, M.D.; Richard K. Helm, M.D.

Board of Censors:

Philip H. Woutat, M.D.; Ralph E. Leigh, M.D.; John H. Graham, M.D.

Corporate Members of the Blue Shield Corporation:

Robert H. DeLano, M.D.; Frank A. Hill, M.D.; Richard H. Leigh, M.D.; William C. Nelson, M.D.; Robert C. Painter, M.D.; William T. Powers, M.D.; Keith G. Vandergon, M.D.; John A. Sandmeyer, M.D.; George A. Wilson, M.D.; Kenneth S. Helenbolt, M.D.; Harold W. Evans, M.D.; William P. Teevens, M.D.; B. J. Clayburgh, M.D.

Representatives to the Board of Directors of the North Dakota Physicians Service:

George L. Countryman, M.D.; Kenneth S. Helenbolt, M.D.; William T. Powers, M.D.; Harold W. Evans, M.D.

Dr. Powers moved that the nominations for the officers of the Third District Medical Society be closed and to cast a unanimous ballot for the above named. Motion was carried.

A similar motion was made by Dr. Silverman and seconded by Dr. Eelkema concerning the delegates to the North Dakota State Medical Association. Motion was carried.

Dr. McLeod moved that Dr. Woutat be elected to the Board of Censors. This was seconded by Dr. Clark and the motion carried unanimously.

The Blue Shield Corporation members and representatives were accepted on a motion by Dr. Helm and seconded by Dr. William C. Nelson. The motion was carried.

Dr. Warner gave a final report of the Diabetes Detection Drive and his report, as well as the financial statement is included with these minutes.

Dr. John Graham gave a report concerning his liaison on the Valley Memorial Home. It was moved by him and seconded by Dr. Clark that the Valley Memorial Home send out letters to physicians who have forgotten to see their patients near the end of the month. This motion was carried.

A letter was read from Dr. Amos, State Health Officer, concerning the availability of INH at a normal cost from the San Haven Sanatorium.

The local druggists have asked our endorsement concerning their program during Poison Control Week. This was moved by Dr. Sandmeyer and seconded by Dr. Silverman that this activity be endorsed.

The meeting was adjourned.

The regular meeting of the Third District Medical Society was held on February 16, 1967. The program consisting of Dr. Bruce Williams of Duluth, Minnesota on "Dysfunctional Uterine Bleeding" was excellently presented and a very, very interesting discussion followed.

The meeting was called to order by President Harold I. Tarpley, M.D., at 8:30 p.m. Dr. Powers moved and Dr. Clayburgh seconded that the minutes be approved without reading and this motion was passed.

The Treasurer's report was given informally. It was noted that there was a balance in the order of several thousand dollars. Dr. Barcome moved and Dr. Haunz seconded that a Committee be appointed to dispose of the excess funds and this motion was passed.

Dr. Powers reported as City Health Officer that beginning March 1, there would be a Home Health Care Program initiated in Grand Forks.

Dr. Tarpley presented as a project for the Third District Medical Society the development of a curriculum for sex education in the schools of the District. This was extremely well received and stimulated much discussion, all of which was favorable. Dr. Clark moved, seconded by Dr. Haunz, that the District Medical Society recommend to the School Board, the development of a curriculum on sex education. Motion was passed. Dr. Sandmeyer moved, seconded by Dr. Haunz, that the President appoint a committee to get a curriculum developed. This motion was passed.

The meeting was adjourned.

The regular meeting of the Third District Medical Society was held at the River Bend Country Club, East Grand Forks, Minnesota, on March 16, 1967. Dr. Ian Smith, Professor of Medicine at the University of Iowa, presented the program on "The Selective Use of Antibiotics" which was well received.

Following dinner the regular business meeting was called to order by the President, Dr. Tarpley, at 8:15 p.m. Dr. Powers moved and Dr. Richard Leigh seconded that the minutes be approved without reading. Motion approved.

The report of the Audit Committee was given by Dr. Harold Evans. It was moved by Dr. Powers, seconded by Dr. Wilson, that the report be accepted. Motion carried.

Committee appointments were announced by Dr. Tarpley as follows:

Committee on Education for Living, William T. Powers, M.D., Chairman.

Program Committee, Duane F. Pansegrau, M.D., Chairman; John A. Lambie, M.D.

The Secretary announced that at the May 19, 1966, meeting, the Society passed a motion to the effect that work would be done at the usual and customary fees. The purpose of bringing this up at this time was to establish a basis for negotiating with the Head Start Program.

The meeting adjourned at 8:45 p.m.

Respectfully submitted,
(Signed) Ralph E. Mahowald, M.D.,
Councillor, Third District

Fourth District

The Northwest District Medical Society (Fourth District) held seven meetings during our fiscal year beginning March 1966 and ending in February 1967. Our meetings this past year have been very well attended and have been held at various local facilities, all providing accommodations that adequately meet our Society needs. As of March 1st, 1967, we have sixty regular members. There are four new members admitted during this past year. They are as follows:

1) Dr. Allardice, Stanley; 2) Dr. Stump, Minot; 3) Dr. Chanco, Powers Lake; 4) Dr. Ilvedson, Minot.

At this time there is also an application pending for membership by Dr. Liu of Minot. We also have two limited members, namely Dr. Peterson who at the present time is serving a residency and Dr. Vandersteen who will soon be leaving for a residency. We have seven Honorary members, these being:

1) Dr. A. R. Sorenson; 2) Dr. A. L. Cameron; 3) Dr. Paul Rowe; 4) Dr. Max Lampert; 5) Dr. M. G. Flath; 6) Dr. G. Roy Ringo; 7) Dr. A. E. Westervelt.

The officers elected for the 1966-1967 year were:

President	W. C. Hurly, M.D.
Vice-President	L. A. Giltner, M.D.
Secretary-Treasurer	B. Jayapathy, M.D.

Our first meeting of the new fiscal year was held on March 31, 1966. A delightful social hour was held at the

new home of Dr. W. B. Huntley, after which our business meeting convened at the Lutheran Home for the Aged in Minot. Dr. O. H. Hove, Administrator of the Lutheran Home, spoke briefly of the effects of the Medicare Law and the operations of the Lutheran Home. It was at this meeting that a resolution was passed, Resolved: That the selection of a councillor be made from a list of candidates given by the individual district societies. Dr. A. M. Cameron discussed a letter from Dean Harwood regarding Public Law 239 setting up regional centers for heart disease, stroke and cancer. Twenty-one members were in attendance.

No meeting was held in April since this meeting would have been held only a few days before our State Medical Meeting. Our May meeting was held at the Minot Country Club on the 19th. The business meeting was called to order by Dr. Hurly at which time I gave my first report as a newly elected Councillor of the Northwest District Medical Society concerning the proceedings of the Council at our recently completed annual meeting. Mr. Donald Eagles, Executive Director of North Dakota Blue Shield then gave a very informative report concerning the negotiations between Blue Shield and the Department of Health, Education and Welfare regarding the administration of Part B of the Medicare Program. Twenty-five members were in attendance.

There were no meetings held in June, July or August. Our next meeting was held on Thursday, September 8th at the Ramada Inn. Our president, Dr. Hurly, introduced our speaker of the evening, Dr. Haunz, who spoke on current concepts of diabetes and its treatment. His presentation was very well received and numerous discussions ensued. A report of the Indian Affairs Committee was given at this meeting. This committee was organized a few months earlier in an effort to work out some problems between the Indian Bureau and physicians of the Northwest District rendering medical services to this group of people.

On Thursday, October 20th, the Northwest District Medical Society met at the Minot Country Club. The meeting was conducted by Vice-President, Dr. Lloyd Giltner, in the absence of our president, Dr. Hurly. Dr. Richardson submitted a report of the meeting of the Medical Economics Committee and the Public Welfare Board primarily dealing with fee schedules. There was a general opinion that the Medical Economics Committee and others had done a splendid job in negotiations with the Public Welfare Board in behalf of the entire State Medical Society. It was also pointed out that the 1966 North Dakota Relative Value Guide would soon be off the press and would be used as a guide in dealings and negotiations with all agencies, both state and federal, in the future. Dr. Huntley discussed the Fee Profile as established by North Dakota Blue Shield. Our speaker for the evening was Dr. O. H. Hove who is Chairman of the Board of Minot Mental Health Committee. The official name of this organization is the Ward County Mental Health and Retardation Service Unit. This unit was made possible by a levy which was passed at the last general election. This unit plans to provide outpatient-inpatient consultation and emergency services and are attempting to secure a qualified psychiatrist. A fee schedule has been submitted to the state. Some dissatisfaction was expressed by members of the district medical society that no physicians had been appointed as a member of the committee forming this organization. The meeting was concluded with a movie shown by Dr. Stump on the radioactive isotopes in the diagnosis of various diseases. Twenty-five members were present.

On November 11, 1966 a meeting of the Northwest District Medical Society was held at the Minot Country Club with Dr. Hurly presiding. The society was saddened at the news of the accident of our Executive Secretary, Mr. Lyle Limond and our secretary was instructed to convey the sympathy of our entire group. Our speaker for the evening was Dr. B. Jayapathy who spoke on "Management of Clefts of the Lip and Palate". It is always gratifying to hear a well presented scientific paper but especially when it is given by one of our own Northwest District Medical Society physicians. Twenty-four physicians were in attendance.

Our next meeting was held on January 26, 1967 at the Minot Country Club. Considerable discussion was given to current bills before the North Dakota Legislature, particularly those relating to the Podiatrists, and to the bill which would have brought Blue Shield under the jurisdiction of the State Insurance Commissioner. The treasurer's report was read showing the District Society to be in sound financial condition. A motion was made and unanimously passed allowing Air Force physicians stationed at the Minot Air Force Hospital to practice medicine in the Minot area to cover the various specialties at times needed. Dr. Larson spoke about the changing patterns of Nursing Education in our country today. He made a motion that the Northwest District Medical Society go formally on record as supporting the principles of a four year degree program for nurses at Minot State College and that towards this end the president of the Northwest District Medical Society should appoint a committee to meet with the State Board of Higher Education to implement such a program. Considerable discussion ensued but the motion was seconded and passed 26-3. Election of officers was then held. The nominating committee consisted of Drs. Richardson, Larson and Kohl. The following were elected:

1) Dr. Larson re-elected to the Blue Shield Board of Directors.

2) Twelve members were nominated to the Blue Shield Corp.

3) Delegates to the State Medical Association: 1) Lloyd Giltner, M.D.; 2) B. Jayapathy, M.D.; 3) A. M. Cameron, M.D.; 4) Gale Richardson, M.D.

The following alternates were elected in the order named: 1) W. B. Huntley, M.D.; 2) J. Boyle, M.D.; 3) S. Shea, M.D.; 4) S. Keck, M.D.

4) The following physicians were elected as officers of the Fourth District Medical Society for the 1967-68 year:

President	Dr. Lloyd Giltner
Vice-President	Dr. Burton Olson
Secretary-Treasurer	Dr. B. Jayapathy

The business meeting was then concluded and our speaker for the evening was Dr. Richard Fardal, Dermatologist at the Quain and Ramstad Clinic in Bismarck. Dr. Fardal presented a very informative and interesting talk illustrated by numerous color slides on some of the common skin disorders.

Our final meeting of the fiscal year was held on February 23, 1967, at the Clarence Parker Hotel, conducted by our new president, Dr. Lloyd Giltner. Dr. Ron Ilvedson was elected to the membership of our Society. Dr. Giltner announced that he had appointed Dr. Larson as Chairman of the Nursing Education Committee and also serving with him on this committee were Drs. Heidorn, Stump and Giltner. Since the last meeting the Committee had met with representatives of both hospitals and with Dr. Lura of Minot State College on two occasions. Considerable discussion of this program again ensued. Concern was expressed over the possibility of

taking steps to insure that no further shortage of nurses could or would develop through our efforts to establish a four year program at MSC. The district society unanimously went on record as continuing to support our diploma schools and that an effort be made to effectively integrate the diploma education program with the newly proposed degree program to insure a continuous supply of nurses. After much discussion the motion was made that the Northwest District Medical Society instruct the special committee appointed by Dr. Giltner to contact the State Board of Higher Education requesting the Board to develop a four year degree nursing program at Minot State College with the additional recommendation that the feasibility be studied further of integrating the proposed four year degree program and the present diploma programs of nursing. The motion was seconded and passed. Dr. Rioux then spoke at some length about Senate Bill 190. Dr. Rioux proposed certain amendments to the bill which in a sense would permit psychotherapy by psychologists only under the direct supervision of a psychiatrist. A motion was then made, seconded and passed unanimously to recommend amending Senate Bill 190 as proposed by Dr. Rioux. The scientific program consisted of a talk on "Experiences with a Medical Intensive Care Unit" given by Dr. Ralph Spiekerman of the Mayo Clinic. An excellent discussion followed his presentation. Thirty-three physician members were in attendance at this meeting.

This has been an eventful year and the members of the Fourth District Medical Society are to be congratulated for their participation and work toward solving the problems confronting us as a Medical Society this past year.

Respectfully submitted,
(Signed) Richard S. Larson, M.D.,
Councillor, Fourth District

Fifth District

The Sheyenne Valley Medical Society held eight meetings during 1966.

The first meeting was held in January, with election of officers and delegates as follows:

President: Milton O. Berg, M.D.; Vice-President: Neil A. Macdonald, M.D.; Secretary-Treasurer: John W. Goven, M.D.; Delegate: Clayton E. Jensen, M.D.; Alternate Delegate: Clifford J. Klein, M.D.; Blue Shield Corporation Members: G. Christianson, M.D., Clayton E. Jensen, M.D.; Director of Blue Shield: Clifford J. Klein, M.D.; Program Chairman: Warren R. Jensen, M.D.

Following the election there was a film on I.P.P.B.; and the uses and techniques of anterior cardiac massage. Also present were several Sisters of Mercy.

The next meeting was held in February, when Lester D. Shook, M.D., spoke on the common x-ray finding in general practice with attention to differential diagnosis. He also gave a summary of the uses of radioisotopes.

At the March meeting Hugh D. Thorfinnson, M.D., spoke on embolic occlusion of vessels in the lower extremities, and treatment using drugs and surgery.

At the April meeting we discussed AMPAC-COMPACT, and reasonable, customary and usual fees. Our guest speaker for the evening was James W. Rosevear of Minneapolis, Field Representative for AMPAC.

Steve Pezalla of the Blue Shield office attended our May meeting. He gave a talk regarding Title XIX, submission of claims, and the responsibility of those concerned with management.

On September 19, 1966, the State Medical Association President, Glenn W. Toomey, M.D., gave an address on the public image of the medical doctor, the current legislation, and the need and use of central organization of the state society. Mr. Lyle Limond, our guest, spoke of Title XVIII and Welfare.

At the October meeting, Robert D. Story, M.D. spoke on the coronary care unit. He also presented slides and information on cardiac arrhythmia.

The Sheyenne Valley Medical Society lost its oldest member, A. C. Macdonald, M.D., through death on November 2, 1966. He had been a practicing physician for over fifty years. His many years of medical experience is a great loss to our profession.

Our next meeting was in November when Glenn Sherwood, Ph.D. of the North Dakota Game and Fish Department gave a talk on the present status and welfare of the Canadian Goose. Guests were Delbert Tibke, local game warden, and Dr. James B. Hoyme, Psychiatrist at the State Hospital, Jamestown, North Dakota.

Respectfully submitted,
(Signed) C. J. Klein, M.D.,
Councillor, Fifth District

Sixth District

I am again dividing my Councillor's report into paragraphs, giving a chronological report of my activities and meetings attended.

I was unable to attend the Council Meeting at Fargo, April 28th to May 1st, 1966, because of illness. Dr. Jim Moses attended in my place.

At the Sixth District Meeting May 18, 1966, I discussed the previous meeting of the Council, reviewing all the reports. Mr. Don Eagles discussed Medicare and said that payments made to physicians would be on the same basis as their usual customary fees. A discussion was held on the recommendations of the Council. The speaker of the evening was Mr. James Rosevear, Field Representative of AMPAC. He urged political activity on a local and national level by all members of organized medicine.

I spoke on "Handling Poison Emergencies", at the state-wide course on Immediate Care of the Sick and Injured, held at the State Capitol, June 22, 1966.

I attended a Chamber of Commerce-City Commission joint session on July 8, 1966, which involved discussing hospital and airport facilities.

At the State Meeting of the American College of Physicians at Minot, September 10, 1966, I moderated one of the sessions.

Attended a meeting of an Ad Hoc Committee for the Civic Center on September 19, 1966.

Attended the dedication of the new Basin-Electric Power Plant on September 24, 1966.

Attended Republican West District Workshop on September 20, 1966.

A meeting of the Sixth District Medical Society was held on October 19, 1966. A necrology report was given, memorializing Doctors Hetzler, Gibson and Anderson. Doctors Chiu, from Turtle Lake, and O'Toole, from Grand Forks, were voted into the Sixth District Medical Society by transfer. Dr. Lindelow was elected to the Blue Shield Board to fill Dr. Cleary's place. Dr. Dahl, in discussing the Blue Shield policy, stated that he felt that the Blue Shield Board needed more direction from the local society. There was some discussion of placing emergency equipment at public places, such as the Memorial Building and the Airport, this to be set up by the Medical Society. Dr. Marlin Johnson gave a report of the last Blue Shield meeting, discussing separate forms, anesthesiologist's fees, assistant surgeon's fees and eventual addition of laymen to the Board.

Dr. Toomey, making his annual visitation to the Society, discussed Medicare, the image of the doctors, state dues and the next Legislative Session.

Attended the Bowman-Haley Dam dedication, on October 15, 1966.

Attended the Annual Meeting of the National Committee on Accident Prevention at the Annual Meeting of the American Academy of Pediatrics, held in Chicago, October 25, 1966.

Organized precinct 51 and worked as Republican Precinct Committeeman during the fall national elections in 1966.

Elected Vice-Chairman of the Bismarck Park-and-Shop Board on November 22, 1966.

The Sixth District Medical Society Annual Meeting was held on December 6, 1966. Officers were elected as follows:

President	
P. R. GREGWARE, M.D.	Bismarck
President-elect	
JOSEPH CLEARY, M.D.	Bismarck
Secretary-Treasurer	
JOHN B. FRANKEL, M.D.	Bismarck
House of Delegates	
O. V. LINDELOW, M.D. Term expires 1967	Bismarck
J. J. MOSES, M.D. Term expires 1968	Bismarck
M. A. K. LOMMEN, M.D. Term expires 1968	Bismarck
R. J. DUNNIGAN, M.D. Term expires 1968	Bismarck
EDMUND VINJE, M.D. Term expires 1969	Hazen
M. J. E. JOHNSON, M.D. Term expires 1969	Bismarck
WILLIAM RIECKE, M.D. Term expires 1969	Bismarck
Alternate Delegates	
ARTHUR EWERT, M.D.	Beulah
W. D. WALDSCHMIDT, M.D.	Bismarck
W. J. ORCHARD, M.D.	Linton
J. P. SAUER, M.D.	Bismarck
P. R. GREGWARE, M.D.	Bismarck
G. E. TOLSTEDT, M.D.	Bismarck
JOSEPH CLEARY, M.D.	Bismarck
Board of Censors	
E. D. PERRIN, M.D. Term expires 1969	Bismarck
P. L. OWENS, M.D. Term expires 1967	Bismarck
R. W. HENDERSON, M.D. Term expires 1968	Bismarck
Board of Directors, North Dakota Physicians Service	
O. V. LINDELOW, M.D. Term expires 1967	Bismarck
M. J. E. JOHNSON Term expires 1967	Bismarck
EDMUND VINJE, M.D. Term expires 1968	Hazen
A. F. SAMUELSON, M.D. Term expires 1969	Bismarck

Attended the Interim Council Meeting at Fargo, December 10, 1966.

The Sixth District Medical Society met on February 7, 1967, and heard an address by Dr. John Harkness, Director of the Heartview Foundation. He stated that Heartview Foundation is a non-profit, non-sectarian organization founded in 1963 by a group of civic leaders throughout the State of North Dakota, who are concerned with the welfare of their fellowmen. It is a privately endowed service, supported entirely by voluntary contributions and fees for services. The doors at Heartview are open to all who feel a need to resolve the problems associated with the use of beverage alcohol. Heartview will provide information, guidance and counsel to individuals and families. The goals of Heartview Foundation are to halt the progression of alcoholism before severe social, family and occupational disintegration has taken place and to restore men and women to positions of dignity and productivity within their family and community.

At this meeting Dr. C. A. Arneson was elected to the Board of Directors of the North Dakota Physicians Service, to replace Dr. Kalnins.

Re-elected Chairman of the Bismarck Medical Foundation on February 14, 1967.

During the years 1966 and 1967 I maintained my activity and interest in AMPAC and COMPACT, serving as Membership Chairman for the Southwestern District.

I again served on the Cancer Committee of the North Dakota State Medical Association, as Secretary of the Bismarck Hospital Medical Staff and as a member of the Executive Committee of the Bismarck Hospital Medical Staff.

I again served on the Committees of Accident Prevention and Juvenile Delinquency of the American Academy of Pediatrics.

In January I was elected to the Board of Directors of the Bismarck Regional Mental Health Association.

The reasons for medicine are well summarized by Howell (J. Pediat. Surg. 1:2, 1966):

1. To fight disease on every front.
2. To prevent untimely, unnecessary or avoidable death.
3. When death is inevitable, to make it bearable.
4. After death, to salvage the family.

Respectfully submitted,
(Signed) Robert B. Tudor, M.D.,
Councillor, Sixth District

Seventh District

Since the last report the following meetings were held of the Stutsman County District Medical Society.

The first meeting was held March 24, 1966. A principal piece of business transacted at this meeting involved the giving of $6,744.10 to the Jamestown College, for use for scholarships for nursing students and also to make available loans to these students. This money had been acquired by the polio drive of several years ago, and had been kept in a bank, at interest. There had been numerous proposals for its use previously but this was the first one accepted by the Society. Dr. Hubert Carbone, Superintendent of the State Hospital, was speaker of the evening and his subject was "Psychotherapy in Medicine". He stated that 50 to 70 percent of patients in a doctors office probably have a problem of a mental or psychological nature. He discussed the nature of anxiety and somatic symptoms related to anxiety.

The wives were present at this meeting, as well, and the program was very well received.

The second meeting was held April 21, 1966. A majority of the membership was present and there were several guests. There was no business brought before the meeting and President, Dr. Carbone, introduced Dr. Phillip Dahl of Bismarck, who spoke on COMPACT-AMPAC, and stated that in spite of the passing of the Medicare Bill, the COMPACT-AMPAC organization still existed. Mr. Rosevear also spoke at the meeting for AMPAC and discussed its aims and functions.

Dr. C. E. Johnson of the Mayo Clinic staff in Obstetrics and Gynecology spoke on the subject of "Chemotherapeutic Agents in the Treatment of Advanced Gynecological Cancer". Dr. Johnson illustrated the talk by slides and discussed the use and effects of chemotherapeutic agents. One important point he made was that due

to PAP Smears, we are not seeing as much advanced carcinoma of the cervix as years ago.

The third meeting was held on May 26, 1966 with the majority of the membership present and there were also three medical students here for preceptorship as guests and several guests from the State Hospital. Dr. Hieb reported to the Society on the actions of the House of Delegates at the State meeting in the first part of May. Dr. Van der Linde, Councillor for the Society, also spoke upon the transactions of the North Dakota State Medical meeting. There were several pleas made for COMPACT-AMPAC and the AMA-ERF. At this meeting the transfer of funds from the Medical Society Treasury to the Jamestown College was consummated and no special restrictions were placed on it, although it was felt that it should be used for nursing education.

The program for the evening consisted of two films on "Nutritional Anemia and Megaloblastic Anemias".

After the summer recess, the fourth meeting was held on October 27, 1966 and many guests were present. Dr. Glenn Toomey, President of the State Medical Association, Lyle Limond, Executive Secretary, Dr. O. A. Sedlak, Medical Consultant for Blue Shield, and Don Eagles, Executive Director of Blue Shield, were all present. The program consisted of Dr. Toomey making a presentation and his basic theme was "The Attitude of the Government Concerning the Medicare Bill". He felt that this was just an initial phase of complete socialization of society in this country. His talk was very thought-provoking and involved many facets of society in general as well as medical practice.

Dr. Sedlak complimented the doctors on trying times that they have had during early Medicare months and stated that the claims had been coming in adequately and that his department had no complaints. The program following consisted of a film on "Hypothyroidism".

The fifth meeting was held November 17, 1966. The majority of the membership was present. Secretary Dr. Nierling mentioned that the dues for the coming year would be $225.00 including the Auxiliary dues and meals for approximately seven meetings a year. A motion was made that a doctor should be present at the local high school football and local college football games. This motion was passed. The speaker for the evening was Dr. Eugene Brasseur, Ophthalmologist at the DePuy-Sorkness Clinic in Jamestown, who spoke on "Useful Drugs in Ophthalmology". He mentioned many commonly used drugs and gave an excellent presentation of the office practice of ophthalmology principally concerned with the external conditions of the eye.

The sixth meeting was held on January 26, 1967. This was the annual meeting at which new officers are elected. A majority of the membership was present and also several guests from the State Hospital. The officers elected were: President—Philip Curtis, M.D.; Vice-President—G. R. Swafford, M.D.; Secretary-Treasurer—R. D. Nierling, M.D.; Delegates to the State Medical Society—Drs. Edwin O. Hieb and J. V. Miles; Alternate Delegates—Drs. Duane Clement and James Bishop. Censors—Drs. Philip B. Curtis, J. N. Elsworth and Bernard March. Membership of the Blue Shield Corporation—Drs. Joseph Sorkness, D. M. Clement, J. V. Miles, John Van der Linde, John A. Swenson, Edwin O. Hieb and T. E. Pederson. Elected as directors of Blue Shield were Drs. Joseph Sorkness and John A. Swenson. The application for membership into the Society by Dr. Raymond Pilot was approved and Dr. Pilot was unanimously voted into membership. The main portion of the evening was devoted to discussion of the State Legislative activities in regard to medical practice. As might be expected, this became a very prolonged and involved discussion.

The seventh meeting of the Society was held March 30, 1967 with the majority of the members present and several visitors from the State Hospital. A discussion was held regarding a sigmoidoscopy clinic to be held in Jamestown. The dates were set for the last two weeks in April.

Discussion about VD control ensued because the Auxiliary is promoting the showing of films to High School students. It was decided that all doctors would be willing to serve if they are called on for discussion of this matter. The scientific program was given by Dr. Les Shook, Radiologist of the Fargo Clinic of Fargo, and Dr. Shook presented many interesting slides of interesting cases in his experience during his work at the Fargo Clinic.

Respectfully submitted,
(Signed) John M. Van der Linde, M.D.,
Councillor, Seventh District

Eighth District

The Officers of the Kotana District for the year 1966 were elected as follows:

President: M. J. Olson, M.D., Williston

Vice-President: D. E. Skjei, M.D., Williston

Secretary-Treasurer: Dean Strinden, M.D., Williston

The District Society held the following meetings:

March 3, 1966: The wives were entertained at this meeting. Dr. J. D. Craven, President of the North Dakota State Association, addressed the group.

April 1966: This was a dinner meeting and the guest speaker of the evening was Dr. A. F. Samuelson of Bismarck.

September 29, 1966: This was a dinner meeting and the wives of the members were entertained. Guest speaker of the evening was Dr. A. Malcolm Cameron of Minot.

October 13, 1966: Dinner meeting. Dr. James O'Toole of the Public Welfare Board, Bismarck, guest speaker of the evening.

December 18, 1966: This was a dinner meeting and the wives of the members were entertained. Dr. Worthington of the Mayo Clinic, Rochester, Minnesota, was the guest speaker of the evening.

February, 1967: This was a business meeting with election of officers: The following officers were elected:

President: Dr. D. E. Skjei, Williston

Vice-President: Dr. Dean Strinden, Williston

Secretary-Treasurer: Dr. Robert Koch, Williston

Delegate: Dr. D. E. Skjei, Williston

Alternate Delegate: Dr. D. C. Oliver, Watford City

The fall of 1966 saw much activity in the field of politics with much interest and activity on the part of physicians on the local and state level.

Dr. D. E. Skjei served as county chairman of the Republican party and there was much interest and support of the AMPAC-COMPACT activity.

Respectfully submitted,
(Signed) Dean R. Strinden, M.D.,
Councillor, Eighth District

Ninth District

The Southwestern District held a total of only 3 meetings within the past year, the first of these being April 9, 1966. At that time we had a discussion by Mr. Donald Eagles on some of the facets of MEDICARE as related

to its overall pattern and its implementation by our local Blue Shield.

On October 8, 1966 the Southwestern District met at the Ray Hotel and heard a paper by a Mr. James Rosevear who is Field Representative for AMPAC. Mr. Rosevear made several interesting points in his discussion about AMPAC which gave the membership insight into the political action being carried out by doctors throughout the country.

On February 4, 1967 three of our colleagues from Fargo braved their way out west to present an afternoon symposium on "Rheumatoid Arthritis". This pioneering group included Drs. Mack Traynor, D. Ross Halliday, and Ann H. Schutt. This was a very interesting and informative session for all the members of our District Society. That evening, we gathered with our spouses to enjoy the hospitality of the President in the Crown Room and despite a brisk storm outside that interfered with the appearance of our guest speaker, State President Glenn W. Toomey, a warm time was had by all.

The officers for the year 1967 elected at the February meeting were as follows:

President	DONALD A. NICELY, M.D.
Vice-President	H. L. REICHERT, M.D.
Secretary-Treasurer	R. F. RAASCH, M.D.
Corporation Members	DRS. R. J. DUKART, R. E. HANKINS, K. G. FOSTER, A. R. GILSDORF, and W. C. HANEWALD
Delegates	DRS. W. C. HANEWALD and R. E. HANKINS
Alternate Delegates	DRS. R. F. GILLILAND and N. B. ORDAHL
Councillors	DRS. A. J. GUMPER, D. J. REICHERT and R. C. THOM

Respectfully submitted,
(Signed) KEITH G. FOSTER, M.D.,
Councillor, Ninth District

Tenth District

The Traill-Steele District Medical Society had its regular meetings and scientific sessions during the past year. As Councillor of the District, I was very pleased with the response of all members when each was called upon for help during the past legislative session. Almost all members responded with telegrams and telephone calls which had a great effect on accomplishing our purposes. I also am pleased with the cooperation of our 20th District legislators for the help that they gave to medicine.

The members of our Society on State Committees have been active and faithful in attending their meetings. Ours is a small Society, but a very active and helpful one for the cause of medicine.

During the past year Dr. Peter Patton of Cooperstown was elected to membership.

At our February 15th meeting, the following officers were elected for the present year:

President	DR. D. J. HLAVINKA
Vice-President	DR. P. PATTON
Secretary-Treasurer	DR. R. W. McLEAN
Delegate	DR. JAMES LITTLE
Alternate Delegate	DR. KENNETH WAKEFIELD
Corporate Members to Blue Shield	DR. JAMES LITTLE and DR. R. W. McLEAN
Board of Directors	DR. R. W. McLEAN
Censor, 3 years	DR. H. A. LaFLEUR
Censor, 2 years	DR. D. J. BREEN
Censor, 1 year	DR. KARL OJA
Councillor	DR. R. W. McLEAN

Respectfully submitted,
(Signed) R. W. McLEAN, M.D.,
Councillor, Tenth District

STANDING COMMITTEES

Committee on Medical Education

The Committee on Medical Education met in Fargo November 20, 1966. The following members of the Committee were present: Drs. Kihle, Howald Hall, Mark Lane, T. H. Harwood, and R. J. Ulmer. The initial item of business was a report from the Dean of the medical school. No difficulty in transferring students to other schools had been encountered and satisfactory arrangements had been worked out with the University of Minnesota in this regard. He stated also that they had been able to maintain faculty salaries at a reasonably satisfactory level although these were still somewhat below the median for the United States. A few instructors were being lost each year, but this was not considered to be an abnormal drain on man power. The attrition rate for medical students during the first two years at Grand Forks remains between 12 and 15 percent which is considered satisfactory as based on statistics available from other schools. The problem of financing the medical school was also brought up and must be concluded from the figures given that the Medical School will need additional appropriations from the legislature in future years to make up for the deficit in the budget not covered by the mill levy. The Committee on Edueation advised the legislative committee and House of Delegates that the Association should support the concept of continuing the mill levy at the present level and asking for supplementary appropriations from the legislature as needed.

The Cancer, Stroke and Heart Disease Program was discussed. The Education Committee recommended to the officers of the Medical Association that an application for a feasibility study for North Dakota be completed as soon as practical.

There was also discussion about the Student Loan Fund, and the changes that were made in the law two years ago. At this time the committee felt that the Medical Association should again seek to have the law amended to eliminate the size of the city for forgiveness and also to include residency training programs in the eligibility requirement.

The proposed two year associate degree program in nursing education was discussed, particularly as related to the plans to establish this type of training at the Dickinson State College and North Dakota State University. The committee recommended to the State Association that they use their influence to promote the training of additional nurses in the state by this means.

The matter providing for a third and fourth year medical education in the state was brought up for discussion. Dr. Lane took exception to the conclusion in the report submitted by the Dean about the practicability of establishing a third and fourth year school. He pointed out that Fargo had a large enough number of beds and enough of the medical specialties covered here to offer a satisfactory third and fourth year medical curriculum. He recognized, however, that the first step towards this would be the establishment of more accredited residency and internship training programs. The committee recommended that the feasibility of this be included in the study that was to be made for the Heart, Stroke and Cancer Programs.

Dean Harwood recommended that a letter of thanks be sent to the Woman's Auxiliary of the State Medical Association for their help with loan funds. The President of the State Association would be asked to send such a letter.

No further business was discussed at the meeting.

Several weeks following the meeting, a request was received from Dr. Tom Pederson, the Delegate to the AMA from the North Dakota State Medical Association, requesting the Education Committee review the Millis report, also known as the Citizens Commission on Graduate Medical Education, and that this committee be prepared to offer recommendations to the House of Delegates prior to their meeting in May so that Dr. Pederson could be instructed as to the wishes of the North Dakota State Medical Association in this regard before the meeting of the AMA House of Delegates in June of 1967. A resume of the recommendations taken from this report was sent to all members of the Medical Education Committee and they were asked to return them to the Chairman so that this could be included in the Handbook for the Delegates. However, an insufficient number of replies were received to give any consensus of opinion by the Education Committee. Therefore, the resume of the recommendations is included as a part of this report for action by the Reference Committee to the House of Delegates.

These recommendations are as follows:

Recommendations of The Citizens Commission
on Graduate Medical Education
Commissioned by the American Medical Association
from
THE GRADUATE EDUCATION OF PHYSICIANS
(The Millis Report)

1. (Page 29) Because educational programs properly differ from one institution to another, we recommend that each medical school faculty and each teaching hospital staff, acting as a corporate body, explicitly formulate, and periodically revise, their own educational goals and curricula. To do so would be a healthy exercise for medical educators and a fundamental step toward the solution of many of their educational problems.

2. (Page 37) Comprehensive care probably best indicates the nature of the medical and health service involved. But comprehensive-care physician is an awkward title.

We suggest that he be called a primary physician. He should usually be primary in the first-contact sense. He will serve as the primary medical resource and counselor to an individual or a family. When a patient needs hospitalization, the services of other medical specialists, or other medical or paramedical assistance, the primary physician will see that the necessary arrangements are made, giving such responsibility to others as is appropriate, and retaining his own continuing and comprehensive responsibility.

3. (Page 48) Graduate Programs for Primary Physicians:

The primary content of graduate education for comprehensive care should consist of medicine, psychiatry, pediatrics, medical gynecology, and preventive medicine. The Commission has five recommendations to make.

First, simple rotation among several services, in the manner of the classical rotating internship—even though extending over a longer period of time—will not be sufficient. Knowledge and skill in the several areas are essential, but the teaching should stress continuing and comprehensive patient responsibility rather than the episodic handling of acute conditions in the several areas.

Second, (Page 50) some experience in the handling of emergency cases and knowledge of the specialized care required before and following surgery should be included.

ATT. No. 3(a)—CSOB

Third, (Page 51) there should be taught a new body of knowledge in addition to the medical specialties that constitute the bulk of the program.

It is difficult to define this body of knowledge, for it is not yet adequately developed. By analogy with biology, it would include the medical counterparts of ecology, evolution, and fundamental theory rather than the specifics of molecular biology, virology, or the physiology of individual organs.

Fourth, (Page 52) there should be opportunities for individual variations in the graduate program.

Fifth, (Page 53) the level of training should be on a par with that of other specialties. A two-year graduate program is insufficient.

4. (Page 60) We recommend that each teaching hospital organize its staff, through an educational council, a committee on graduate education, or some similar means, so as to make its programs of graduate medical education a corporate responsibility rather than the individual responsibilities of particular medical or surgical services or heads of services.

5. (Page 62) We recommend that the internship, as a separate and distinct portion of medical education, be abandoned, and that the internship and residency years be combined into a single period of graduate medical education called a residency and planned as a unified whole.

6. (Page 63) We recommend that state licensure acts and statements of certification requirements be amended to eliminate the requirement of a separate internship and to substitute therefor an appropriately described period of graduate medical education.

7. (Page 65) We . . . recommend that graduation from medical school be recognized as the end of general medical education, and that specialized training begin with the start of graduate medical education.

8. (Page 70) We recommend that hospitals experiment with several forms of basic residency training, and that the specialty boards and residency review committees encourage experimentation by interpreting liberally those statements in the residency requirements that now inhibit this form of educational organization.

9. (Page 71) We recommend that the specialty boards, in amending their regulations concerning eligibility for examination for certification, not increase the required length of residency training to compensate for dropping the requirement of a separate internship. This can be done by retaining present wording concerning length of residency training and deleting statements concerning internship training.

10. (Page 73) We recommend that programs of graduate medical education be approved by the residency review committees only if they cover the entire span from the first year of graduate medical education through completion of the residency. (This does not mean that each teaching hospital should be required to offer programs in all specialties.)

ATT. No. 3(b)—CSOB

11. (Page 74) We recommend that programs of graduate medical education not be approved unless the teaching staff, the related services, and the other facilities are judged adequate in size and quality, and that, if these tests are met, approval be formally given to the institution rather than to the particular medical or surgical service most directly involved.

12. (Page 85) We recommend that staff members of university medical centers and other teaching hospitals explore the possibility of organizing an intensive effort

to study the problems of graduate medical education and, where such development appears feasible, they seek to arrange for the development of improved materials and techniques that can be widely used in graduate medical education.

13. (Page 100) We . . . recommend that a newly created Commission on Graduate Medical Education be established specifically for the purpose of planning, coordinating, and periodically reviewing standards for graduate medical education and procedures for reviewing and approving the institutions in which that education is offered.

14. (Pages 104-105) In summary, we recommend that the Commission consist of ten members, all of whom are appointed by the Council on Medical Education; that three of the members be appointed from a list of six or more names submitted by the Association of American Medical Colleges; that two be appointed from a list of four or more names submitted by the National Academy of Sciences; that the other five be appointed by the Council on Medical Education without restriction as to the source of the suggestion; that other organizations involved in graduate medical education be consulted in preparing the lists of names from which appointees are selected; that in later years, successors to the original members be selected from lists of suggestions developed and submitted in the same manner as the lists from which the original members were selected. We recommend that not less than two of the members serving at any one time come from outside the field of medicine; that all of the members serve as individual statesmen of medical education rather than as representatives of particular organizations; and that members be appointed solely on the basis of established records of distinguished service in medical education, the related sciences, higher education, or public affairs.

15. (Page 109) We . . . recommend that each residency review committee include a few members from outside of the particular specialty.

ATT. No. 3(c)—CSOB

Respectfuly submitted,
(Signed) Robert J. Ulmer, M.D., *Chairman*
Committee on Medical Education
R. C. Painter, M.D., *Vice-Chairman*

Committee on Necrology and Medical History

I know not what the future hath
of marvel or surprise,
Assured alone that life and death
His mercy underlies.
And so beside the silent sea
I wait the muffled oar;
No harm from Him can come to me
on ocean or on shore.

Whittier

ARNOLD E. HETZLER, M.D.

Dr. Arnold E. Hetzler, 64, a longtime Mandan practicing physician, passed away unexpectedly on May 12, 1966, in the Mandan Hospital, after one day's illness and hospitalization.

Born October 24, 1901 at Sisseton, South Dakota, Dr. Hetzler came to North Dakota with his parents as a child and received his early edueation at Danzig. He attended Ellendale State College and taught school for three years before entering the medical profession. He studied medicine at the University of Minnesota, graduating in 1929. Dr. Hetzler entered into the practice of medicine at Richardton where he remained for three years. He moved his practice to Mandan in 1933 and had been in practice there until the time of his death.

He was a member of the Presbyterian Church, Elks, Rotary, North Dakota State Medical Association, American Medical Association and related societies.

He married Anne McDonald at Ellendale on May 14, 1929. She survives as do three sons.

FRITZ B. SCHLEINITZ, M.D.

Dr. F. B. Schleinitz, 54, physician and surgeon in Hankinson for 19 years, passed away unexpectedly at his home on June 12, 1966.

He was born December 1, 1911 at Meadowlanes, Minnesota. He graduated from the University of Minnesota with a Doctor of Medicine degree in 1935 and practiced medicine for several years at Battle Lake, Minnesota. After serving with the Armed Forces in the South Pacific in World War II, Dr. Schleinitz moved to Hankinson in 1947. With the exception of the few months prior to his death when illness forced him to temporarily halt his practice, he had served the area for 19 years.

Dr. Schleinitz was an active member of the Hankinson Hospital Committee, an organizational group instrumental in securing a hospital for Hankinson.

He was married to Adele Bruce of Fergus Falls, Minnesota, who survives him as do a daughter and a son.

THOMAS FLOYD GIBSON, M.D.

Dr. Thomas F. Gibson, 41, Mandan, North Dakota, died unexpectedly at his home on July 22, 1966.

He was born November 2, 1924 at Oldham, England and received his medical education at the Manchester University Medical School and the University of Dublin, Ireland. He held numerous hospital posts in England and in 1954 spent a year in the Merchant Navy as a Ship's Surgeon.

In 1957 Dr. Gibson emigrated to Canada and practiced in Saskatchewan until 1962. At that time he wrote the examinations given by the North Dakota State Board of Medical Examiners, and having been granted a license, located at Mohall, North Dakota where he practieed until January 1964. At that time he moved to Mandan, North Dakota where he practiced until the time of his death.

Dr. Gibson was interested in amateur radio and obtained his operator's license in Canada. He also held a private pilot's license in Canada and the U.S.A., and was a member of the Midway Flying Club, Bismarck.

He married Dr. Shellagh Milligan May 31, 1957, in Canada, and she was associated with him in the practice of medicine from that time on. She survives as does his mother, Mrs. Bessie Gibson, Southport, England.

AUGUST F. JENSEN, M.D.

Dr. August F. Jensen, 72, a retired Ophthalmologist, who was credited with contributing hundreds of hours to the Sight Conservation Program of Lions International, passed away on September 25, 1966 in Mesa, Arizona, following a year's illness.

He was born September, 1894, in Menomonie, Wisconsin and moved to the Westby area as a child with his parents where they lived on a farm. He received his medical degree from Northwestern University of Chicago in 1925. Dr. Jensen began his practice at Willow City in 1925, moving to Rugby in 1926 where he lived and practiced until 1936 before continuing his education. His graduate work included study at the University of Pennsylvania in 1936-1937 and a residency at the Eye and Ear Infirmary at the same university until 1938. Upon completion of his residency, he moved to Grand

Forks where he was in practice and made his home for 25 years. He retired in 1964 and moved to Mesa, Arizona.

Dr. Jensen was a member of the American Academy of Ophthalmology, A.M.A., and the North Dakota State Medical Association. He was a World War I veteran, member of the American Legion, Disabled American Veterans, Arcadia Lodge A.F. & A.M., Order of Mystic Shrine—Kem Temple at Grand Forks. He was also a long-time member of the Lions International Club and was a past president and 25-year member of the Lions Club in Grand Forks. He was active in Boy Scouts of America who awarded him the Silver Beaver and Silver Antelope.

Dr. Jensen married Agnes Backen at Heimdal, North Dakota, on June 16, 1929. She survives, as do three sons.

FREEDOLPH E. ANDERSON, M.D.

Dr. F. E. Anderson, 66, a longtime Underwood physician and McLean County Coroner since 1957, passed away in a Bismarck Hospital on October 8, 1966.

He was born July 4, 1900, at Dalbo, Minnesota. He attended high school at Bethel Academy in St. Paul and received his medical education at the University of Minnesota, graduating in 1931. Dr. Anderson came to Underwood at that time and had practiced there since.

A member of the Augustana Lutheran Church, he was active in church, school and civic affairs. He belonged to the Elks Lodge and was a past master of Fellowship Masonic Lodge 122 at Washburn. He served as director of North Dakota Health Care, Inc.

He married Olga Mayer September 14, 1932, at Valley City. She preceded him in death on October 25, 1960. Survivors are two daughters and two sons.

CHARLES M. GRAHAM, M.D.

Dr. Charles Graham, 53, passed away in a Grand Forks Hospital October 31, 1966, where he had been a patient for three weeks.

He was born August 8, 1913, at Crookston, Minnesota, and in 1932 was graduated from Central High School. He received his B.A. and B.S. Degrees in Medicine from the University of North Dakota. He graduated from Northwestern Medical School in 1938 and interned at Henrotin Hospital in Chicago from 1938 to 1939. Dr. Graham practiced at Altona, California, from 1939 to 1941. After serving with the U.S. Army Reserve for two years, he went on active duty, serving in the Army Medical Corps in the South Pacific and was awarded the Purple Heart and the Bronze Star. He retired from active duty in 1945, continuing as an officer in the Army Reserve and retired as a Colonel in 1961.

Dr. Graham was City Health Officer of Grand Forks from 1948 until the time of his death. He also served as State Rehabilitation consultant and Old Age and Survivors Insurance consultant. He was a member of the North Dakota Diabetes Association, a past president of the Grand Forks District Medical Society, member of the North Dakota Medical Association, AMA, American Academy of General Practitioners and the American Association of Physicians and Surgeons. He was also a member of the Kem Temple of the Shrine, Disabled American Veterans, Veterans of Foreign Wars, American Legion, Masons and the Elks.

Dr. Graham was married to Elizabeth Raymond on June 4, 1939. She survives him as do two daughters, three sons, a sister and a brother—Dr. John H. Graham of Grand Forks.

ALEXANDER C. MACDONALD, M.D.

Dr. A. C. Macdonald, 81, a prominent Valley City physician who shunned publicity but became one of the state's most honored professional men, passed away November 2, 1966, in a Valley City hospital.

He was born January 22, 1885, on Manitoulin Island, Ontario, Canada. He moved with his family that same year to Cavalier County in what is now North Dakota where his father homesteaded a farm and tree tract. He attended schools in Hannah, Langdon and Lidgerwood. His medical education was received at the University of North Dakota and Rush Medical School, graduating in June 1914. He interned at Childrens Memorial Hospital in Chicago.

Dr. Macdonald began his medical practice in Fingal, North Dakota in 1914 and remained there until 1918 when he was commissioned and called to active duty with the Army Medical Corps in World War I. Following his discharge in 1919, he moved his practice to Valley City where he resided until his death. He was appointed Valley City Health Officer in 1938 and served continuously in that capacity.

On May 2, 1964, Dr. Macdonald was honored by the North Dakota Medical Association when he was given his 50-Year Pin. Later that same year the Valley City Chamber of Commerce sponsored a recognition dinner for his 50 years of service to Barnes County.

He was an active member of local, state and national medical associations, the U. S. Public Health Association, the American Association of Railway Surgeons, and other related societies. He was a member of the Elks, Disabled American Veterans, American Legion, the Forty and Eight, World War I Veterans and the Half-Century Club at the University of North Dakota.

He was married to Mabel Nayes on May 8, 1918, at Minneapolis, Minnesota, who survives him as do three daughters and one son, Dr. Neil, who was associated with his father in the Macdonald Clinic.

H. ANGUS BOWES, M.D.

Dr. H. A. Bowes, 55, psychiatrist, passed away unexpectedly at his home in Grand Forks on December 6, 1966.

He was born September 9, 1911, at Newcastle-on-Tyne in northeastern England. He was educated at Durham School, Durham University and Kings College of Medicine in England. He later took psychiatric training and became a certified specialist in 1946.

Dr. Bowes worked for four years as a psychiatrist at St. Mary's Hospital, Stannington, England. In 1950 Dr. Bowes went to Canada and was Director of Clinical Psychiatry and Director of Research for eight years at Ste. Anne de Belevue Veterans Hospital in Montreal. He was also a lecturer in Psychiatry at McGill University. In 1958, he accepted a position as director of the first psychopharmacologically oriented Mental Health Center at Aberdeen, South Dakota, where he remained until 1961 when he assumed the position as Director of the Institute of Neurology and Psychological Medicine in Grand Forks.

Dr. Bowes was a Fellow of the American Psychiatric Association and had published a number of articles on research in psychiatry in medical journals and numerous papers on psychopharmacology. He was a member of the Sir Thomas Beecham Society and his hobbies were music, literature and travel.

He was married to Kathleen Bestford on August 14, 1944, at Durham Cathedral in England. She survives him.

EARL B. WEIBLE, M.D.

Dr. Earl Weible, 86, who came to Dakota Territory in 1882 as a child, passed away in Fargo on February 8, 1967. Prior to his death, he had lived at Barnesville, Minnesota for more than a year.

He was born at Jamestown, New York on December 7, 1880. He attended the University of Minnesota and was graduated from the Baltimore Medical College in 1910 and was licensed in North Dakota in 1911. He practiced at Berthold, North Dakota for many years before moving to Abercrombie where he was in practice for 25 years. On October 31, 1963, Dr. Weible was honored by the North Dakota State Medical Association, when he was given his 50-Year Pin.

Dr. Weible came from a family of five brothers. Four of the brothers entered the professions of medicine or dentistry and the fifth brother became a professional writer for newspapers and magazines.

All his brothers have preceded him in death. Only survivors are descendents of his late brother, Dr. Ralph Weible.

Respectfully submitted,
(Signed) E. H. Boerth, M.D., *Chairman*
Committee on Necrology and
Medical History

Committee on Public Health

In regard to the Public Health Committee of the North Dakota State Medical Association, of which I am Chairman, we have had no meetings during the year 1966.

Respectfully submitted,
(Signed) Percy L. Owens, M.D., *Chairman*
Committee on Public Health

Committee on Public Relations

We attended the A.M.A. P.R. Meeting in Chicago in August. The meeting was predominantly concerned with elections and legislation. The national feeling tends more toward Doctor-Patient P.R. than the Madison Avenue type.

Another feature that keeps cropping up is there will be no good Public Relations without very good intra-Professional Relations.

An attempt was made to have a meeting of the Committee during the year; however, we were unable to attain a quorum.

I would like to ask the House of Delegates to consider combining the Committee on Legislation and the Public Relations Committee into a Committee of Public Policy and Legislation. Every two years these Committees have functions almost indistinguishable from each other—the other year one Committee could pretty well handle the situation.

Respectfully submitted,
(Signed) W. M. Buckingham, M.D., *Chairman*
and
F. A. DeCesare, M.D., *Vice-Chairman*

Committee on Official Publication

There was no meeting of the Committee on Official Publication during the past year.

It is recommended that this Committee be dissolved because of lack of function.

Respectfully submitted,
(Signed) Joseph W. Cleary, M.D., *Chairman*
Committee on Official Publication

SPECIAL COMMITTEES

Committee on Cancer

I have made several attempts during the past two years to initiate meetings in conjunction with the State Medical and Interim Meetings. This has proven rather difficult as we have not had important business on our Agenda that requires a meeting. We have decided that, in the future, our Cancer Committee could act as an advisory group on any pertinent cancer matters that would require an opinion or decision. In the future, all members will be contacted by mail, unless an emergency arises.

At the annual meeting of the North Dakota Division of the American Cancer Society in Fargo on September 19, 1966, Doctor Leonard Larson presented a plan to establish a central Cancer Registry for North Dakota, parts of northern South Dakota, and eastern Montana. The cost of operating this Registry would be financed entirely by the Quain and Ramstad Clinic of Bismarck, North Dakota. This met with the approval of the Board of Directors of the North Dakota Division. Members of the State Committee on Cancer were contacted and also approved this plan. You recall, that in 1959, our Committee presented a plan to the House of Delegates to establish a Central Cancer Registry with the Department of Health in Bismarck. This request was not approved. We feel that most of the doctors in North Dakota will approve of this plan. Central Registries have been established in most of the other states of the union.

Feel free to write to the North Dakota Division of the American Cancer Society's office in Fargo if you wish any literature or films for your meetings. Melvin Van Beek, Executive Director, has been granted a leave of absence for one year for added training in New York, but a substitute director will be in charge during his absence.

Respectfully submitted,
(Signed) C. M. Lund, M.D., *Chairman*
Committee on Cancer

Committee on Nursing Education

There was no interest evidenced in the committee either from inside the Medical Society or from the outside.

I would recommend that this committee be abolished as a special committee. Should need arise in a special situation involving nursing education, then the President already has the power to appoint a special committee.

Respectfully submitted,
(Signed) J. H. Mahoney, M.D., *Chairman*
Committee on Nursing Education

Committee on Maternal and Child Welfare

The Maternal and Child Welfare Committee of the State Medical Association met in Fargo and made the recommendation that the present abortion law be changed to include a pregnancy as a result of an incestuous act or rape. This was turned over to the Legal Department of the State Medical Association and the following was drafted by the Legal Department:

"A BILL—For an Act to amend and reenact sections 12-25-01, 12-25-02, 12-25-04 and 12-25-07 of the North Dakota Century Code relating to miscarriage or abortion, and providing penalties.

BE IT ENACTED BY THE LEGISLATIVE ASSEMBLY OF THE STATE OF NORTH DAKOTA:

SECTION 1. AMENDMENT.) Section 12-25-01 of

the North Dakota Century Code is hereby amended and reenacted to read as follows:

12-25-01. PROCURING AN ABORTION—PUNISHMENT.) Every person who administers to any pregnant woman, or who prescribes for any such woman, or who advises or procures any such woman to take any medicine, drug, or substances, or uses or employs, or procures or advises the use, of any instrument or other means whatever, with intent thereby to procure the miscarriage of such woman, unless the same is necessary to preserve her life, *or unless the woman became pregnant as a result of an incestuous act or rape,* shall be punished by imprisonment in the penitentiary for not less than one year nor more than three years, or in a county jail for not more than one year.

SECTION 2. AMENDMENT.) Section 12-25-02 of the North Dakota Century Code is hereby amended and reenacted to read as follows:

12-25-02. ABORTION—IF MOTHER OR CHILD DIES—PUNISHMENT.) Every person who administers to any woman pregnant with a quick child, or who prescribes for such woman, or who advises or procures any such woman to take any medicine, drug, or substance whatever, or who uses or employs, or procures or advises the use, of any instrument or other means with intent thereby to destroy such child, unless the same shall have been necessary to preserve the life of such mother, *or unless the mother became pregnant as a result of an incestuous act or rape,* in case the death of the child or of the mother is produced thereby, is guilty of manslaughter in the first degree.

SECTION 3. AMENDMENT.) Section 12-25-04 of the North Dakota Century Code is hereby amended and reenacted to read as follows:

12-25-04. SOLICITING OR SUBMITTING TO ATTEMPT AT ABORTION—PUNISHMENT.) Every woman who solicits of any person any medicine, drug, or substance whatever and takes the same, or who submits to any operation or to the use of any means whatever, with intent thereby to procure a miscarriage, unless the same is necessary to preserve her life, *or unless the woman became pregnant as a result of an incestuous act or rape,* shall be punished by imprisonment in the county jail for not more than one year, or by a fine of not more than one thousand dollars, or by both such fine and imprisonment.

SECTION 4. AMENDMENT.) Section 12-25-07 of the North Dakota Century Code is hereby amended and reenacted to read as follows:

12-25-07. ABORTION—TESTIMONY OF PERSON INJURED MUST BE CORROBORATED.) Upon a trial for procuring or attempting to procure an abortion, *or miscarriage,* or aiding or assisting therein, the defendant cannot be convicted upon the testimony of the person upon whom the abortion was performed unless her testimony is corroborated by other evidence. *A pregnancy purportedly caused through incest or rape shall not be a defense under the exceptions of sections 12-25-01, 12-25-02, or 12-25-04 unless such fact had been determined by a district judge of the county wherein the alleged incest or rape occurred after consultation by such judge with a medical advisory committee appointed by the president of the district medical society of said county, and unless such consultation and determination were made and recorded in the district court records prior to the alleged violation of sections 12-25-01, 12-25-02, or 12-25-04, and said judge entered his order permitting medical procedures to incur the miscarriage or abortion".*

(The italics indicate the insertion of amendments to said section of existing code.)

This was then sent out to the State Ob-Gyn Society where it was approved by almost 2 to 1 margin and was submitted to the House of Delegates for their consideration.

Respectfully submitted,
(Signed) Robert E. Lucy, M.D., *Chairman*
Committee on Maternal and Child Welfare

Committee on Foreign Trained Physicians

The influx of foreign medical graduates into the United States continues at a constant rate and represents the greatest migration of professional people in history. The number has increased each year since 1951, except for two. To show the magnitude of this problem, it is noted that 27,804 foreign graduates have been licensed in the United States since 1946, and 2,403 were licensed in the past year, with a mortality rate around 32%.

The countries which send us the greatest number of physicians are, unfortunately, those who actually have no physicians to export; namely, countries of the Near East, Far East and Latin America.

Seventy-five per cent of the foreign graduates examined in 1965 were from seven countries; namely, Cuba, the Philippines, Turkey, Italy, Argentina, Greece, and Mexico. The failure rate varied from 24% to 50%.

The State Boards which license the greatest number of foreign graduates are New York, New Jersey, Connecticut, Maryland, Pennsylvania, Virginia, Michigan, Illinois, and Florida.

In 1965, the failure rate for graduates of U. S. Medical Schools was 2.3%; for Canadian graduates, 5.8%; and for foreign graduates, 32.1%

The United States now has 84 Medical Schools, all Grade A, which graduated about 8,000 physicians in 1966. Even so, there is a definite shortage and not enough to staff the approved Internships and approved Residencies in the United States. As a result, there are approximately 13,000 foreign graduates serving as interns and residents.

Last year, there were 14,178 approved Internships, but only 7,753 approved students who were matched to fill these.

The entire internship program is now undergoing close study and evaluation by the Council of Medical Education of the AMA, and there is a possibility there will be drastic changes; such as the elimination of the rotating internship and the development of more straight internships. The elimination of the internship is also under study.

One of the problems associated with foreign graduates is language difficulty. An English language test is now included in the ECFMG examination. Last year, the failure rate in this test was 8%.

Hospital Administrators are assisting in providing means of improving the command of the English language by all of their foreign graduate interns.

It is recognized that all approved hospitals should accept at least a few foreign graduates and that a hospital with an all foreign graduate intern staff is in constant trouble.

SITUATION IN NORTH DAKOTA:

There are approximately 50 foreign graduates licensed and practicing in North Dakota, about equally divided between a specialty and general practice. During the past year, twelve were examined and two failed. This failure rate is considerably less than in the United States as a whole. Since 1946, 75 foreign graduates have been licensed by examination and 2 by reciprocity in North

Dakota. Canadians are not considered as foreign graduates.

DATA:

1. Number of Medical Schools in the United States—84.
2. Number of Medical Schools in Canada—12.
3. Number of Medical Schools in the rest of the world—275.
4. Foreign Graduates in the United States as Interns—2900, plus.
5. Foreign Graduates in the United States as Residents—8200, plus.
6. States which do not license Foreign Graduates—Arkansas, Louisiana, Nevada.
7. Last year, 17,812 physicians were licensed in the United States by 54 Boards. 6,907 by written examination and 10,907 by endorsement.
8. In 1965, 2,043 Foreign Graduates were licensed and 968 failed in their examination. Mortality rate, 32%
9. Approximate number of Physician deaths in the United States each year, 3,800.
10. An ECFMG Certificate is required by all Licensure Boards except 10.
11. The National Board of Medical Examiners endorsed 5,481 Candidates in 1966.
12. 63955 foreign graduates have written the ECFMG examinations since the first one was given in 1958. 63% of these have ultimately received a standard certificate.
13. During the past year about 1700 candidates were taking the examination for the first time.
14. It must not be assumed that passing the ECFMG examinations means the same as passing the National Board examinations. The latter require 5 days of writing while the ECFMG Examination is only one day.
15. 1343 physicians have written the ECFMG examinations in England in the past 6 months. This large number is apparently due to concern over the National Health Service in England and many British physicians are migrating to other parts of the world.

Respectfully submitted,
(Signed) C. J. Glaspel, M.D., *Chairman*
Committee on Foreign Trained Physicians

Committee on Diabetes

Members of Committee: Doctors: E. A. Haunz, Chairman, Grand Forks; Donald M. Barnard, Fargo; B. Hordinsky, Drake; Warren R. Jensen, Valley City; E. O. Hieb, Jamestown; W. H. Wall, Wahpeton; G. H. Heidorn, Minot; Keith G. Foster, Dickinson; P. F. Klosterman, Bismarck.

The Committee on Diabetes, initiated by Dr. Leonard Larson of Bismarck, has continued its primary function to encourage and coordinate annual diabetes detection drives throughout the state under sponsorship of constituent local medical societies. This function is a cooperative effort in support of National Diabetes Week, sponsored in November each year by the Committee on Detection and Education of the American Diabetes Association, Inc. Increasing participation is in evidence among district medical societies throughout the nation. Last year, well over twelve hundred societies held individual detection drives.

Dr. Robert C. Warner of Grand Forks was Chairman of the Diabetes Detection Drive held in that community the week of November 20, 1966, which coincided with National Diabetes Week. This drive was sponsored by the Third District Medical Society with the cooperation of the North Dakota State Health Department. Other members of the committee were Drs. Lloyd S. Ralston, James A. Leigh, Curtis W. Sande, Norman M. Helgason, E. A. Haunz, W. E. Cornatzer, and William P. Teevens. In accordance with the recommendations of the American Diabetes Association, the testing method used was the Drey-Pak glucose oxidase system. The Drey-Paks were distributed during National Diabetes Week in Grand Forks through the banks, the major department stores, the pharmacies, and the University. They were distributed largely through pharmacies in the outlying areas of the district. Following the onset of the Detection Drive, two weeks were allowed to receive the returned Drey-Paks for processing. For personnel reasons at the University where the Drey-Paks were processed, this period could not be extended further.

The funds for this drive were made available through both the Third District Medical Society and the State Health Department. A total of $500 was allocated. Of this, expenditures are approximately as follows: $200 plus transportation costs from New York for the test materials, $40 in clerical expense, and $10 in postage and miscellaneous.

The results obtained were as follows: Out of the Drey-Paks distributed a total of 974 were utilized by the populace and returned for testing. Out of this group the actual number tested was 912. The balance were not tested because of the fact that they were received too late to be included. Out of the group of 912 which were tested, there were 30 positive results obtained. The persons who obtained a positive result were notified by mail and advised to consult with their own physicians relative to further testing to determine whether or not diabetes existed. Those persons submitting Drey-Paks too late to be included in the test program were so advised, and it was suggested to them that they consult their local physicians if they had some cause for serious concern or alternately participate promptly in the next detection drive. Those persons with a negative result were not notified individually but were notified by means of a notice in the Grand Forks Herald to the effect that if they had not received advice to the contrary, they could consider their tests negative.

The State Health Department has repeatedly voiced its interest in participating in diabetes detection in cooperation with the North Dakota State Medical Association and interested physicians throughout the state and gave us excellent cooperation in the 1966 Diabetes Detection Week. It is hoped that in the future a joint cooperative effort may be made with the State Health Department to carry out diabetes detection programs on a larger scale and more efficiently in the future. However, the committee feels that it is most important that the medical profession throughout the state try to maintain the initiative and leadership in this overall project. In fact, diabetes detection drives throughout the United States have been almost exclusively carried out under the sponsorship of local medical societies. This not only stimulates more enthusiasm among participating physicians but sustains the good will which has been engendered toward the medical profession in carrying out this free service to the public throughout the past decade.

This committee sincerely hopes that the constituent medical societies of the North Dakota State Medical Association will give serious thought to carrying out some sort of diabetes detection program in 1967. The expense involved in these drives is minimal because of the cooperative effort of various pharmaceutical manufacturers in furnishing free testing material and the American Diabetes Association, Inc., is glad to cooperate in furnishing publicity materials to assure a uniformly ethical

approach throughout the different projects. Obviously local medical societies are expected to make at least a token contribution toward expenses for an individual detection drive.

I would again like to express my appreciation for the continued excellent cooperation from committee members throughout the year who stand ready to cooperate at any time detection programs are initiated by individual groups.

Respectfully submitted,
(Signed) E. A. Haunz, M.D., *Chairman*
Committee on Diabetes

Committee on Crippled Children

The Committee on Crippled Children held a business meeting Saturday, December 10, 1966 at the Oak Manor Motel, Fargo, North Dakota. The meeting commenced at 10:30 a.m. with Dr. Paul L. Johnson, Chairman, presiding.

Present were: Doctors H. P. Smeenk, James V. Miles, Jr., Clifford Hogan and B. A. Mazur and Myrtle Kohler, Recording Secretary.

In his opening remarks, Dr. Johnson advised the members that during the first years of Crippled Children's Services the cases approved for treatment were mainly orthopedic. He cited a report from the Public Welfare Board of North Dakota that in terms of the number of cases approved for treatment, the diagnoses of greatest frequency in the past seven years have been strabismus, cleft lip and/or palate, congenital malformations of the circulatory system (congenital heart disease), and cerebral palsy. Children and youth approved for treatment of these four conditions constitute slightly less than one-half of the Crippled Children's Services caseload, with one-fifth of the current caseload being approved for treatment of strabismus. Doctor Johnson stressed that, if proposed federal regulations are passed by the Legislature they will be more stringent than the present CCS program, and more rigid than Public Welfare standards are now. It was pointed out to the members of the Committee that CCS is now working on state funds.

In discussing parts of Title 19 in connection with CCS it was the consensus of the group that perhaps general practitioners would handle more cases in the future with specialists consulted to concur with their diagnosis. Dr. Miles said that the cases submitted have always been covered and have always been approved. Dr. Johnson said that, in his opinion, many cases were approved too liberally and that the caseload has increased over the years. He added that restrictions must be more severe to effectively reduce the case load. It was pointed out to the members that the present Relative Value Guide should be acceptable to all physicians. There are still some physicians who do not conform and do not participate in the program. They give care privately with no obligation, handling these cases as general welfare.

After considerable discussion by the members of the Committee, the following statement was presented to the Council after adjournment of the Committee meeting. The Council suggested that this statement be referred to the Committee on Medical Economics for their evaluation.

STATEMENT

The Crippled Children Committee is to go on record as approving the philosophy of providing the best medical services possible for children by the continuation of present Crippled Children's Services and extensions of aid to the medical indigent within the fiscal limitations of providing an adequate but conservative program.

This statement was seconded by Dr. Mazur and passed.

Briefly discussed was the limited program being developed by Dr. Cameron in Minot, North Dakota that partially reduces the strain on out-of-state facilities. Cardiac catherizations are being performed there and diagnosis arrived at, but no surgery is being done at the present time.

There being no further business, the meeting adjourned at 12:30 p.m.

Respectfully submitted,
(Signed) Paul L. Johnson, M.D., *Chairman*
Committee on Crippled Children

Committee on Mental Health

The Committee on Mental Health has had no meetings since the last State Medical Meeting, therefore we have no report.

Respectfully submitted,
(Signed) Paul J. Beithon, M.D., *Chairman*
Committee on Mental Health

Committee on Aging and Rehabilitation

Because of weather conditions the meeting of this committee on December 10, 1966, in Fargo, was poorly attended. A quorum was not present. The chairman prepared a report on the Rehabilitation Conference of the American Medical Association held in Chicago in September, 1966. This report has been filed with the minutes of the meeting.

The other activity of this committee during the year was the co-sponsorship with the North Dakota Heart Association of a film, "Candidate for a Stroke," preceded and followed by discussion of the stroke problem by the Chairman of this Committee. This one-half hour program was presented by the Columbia Broadcasting Company Television Network throughout North Dakota on a Sunday afternoon. Many North Dakotans saw this program. Many favorable and unfavorable comments were subsequently received.

Because of his appointment to this Committee, the Chairman was appointed to the Governor's Policy Board for Statewide Planning for Comprehensive Vocational Rehabilitation Services. Undoubtedly many other members of this committee will also become involved in this program.

Respectfully submitted,
(Signed) Harold W. Evans, M.D., *Chairman*
Committee on Aging and Rehabilitation

Liaison Officer to the North Dakota State Bar Association

No contact was held officially with the representatives of the North Dakota State Bar Association during the past year and no apparent problems were brought to the attention of this individual.

Respectfully submitted,
(Signed) Paul L. Johnson, M.D.
Representative

Committee on Liability Insurance

The Committee on liability insurance has not met this year and has heard no problems needing its attention. This Committee does not feel that it should be concerned with individual medico-legal problems, but only with proper liability coverage, which we believe is available.

Respectfully submitted,
(Signed) Joseph Sorkness, M.D., *Chairman*
Committee on Liability Insurance

Committee on Constitution and By-Laws

Two attempts were made in the year 1967 to obtain a quorum for the meeting of the Committee on Constitution and By-Laws and another attempt will be made between now and the annual meeting.

If we are successful in conducting some business at this meeting, a supplemental report will be made ready for distribution at the time of the annual meeting.

Respectfully submitted,
(Signed) KEITH G. FOSTER, M.D., *Chairman*
Committee on Constitution and By-Laws

Liaison Committee to the North Dakota Dental Association

There was no special activity on the part of the Liaison Committee to the North Dakota Dental Association during the past year.

Respectfully submitted,
(Signed) DAVID G. JAEHNING, M.D.
Representative

Committee on Cardiovascular Diseases

The Committee on Cardiovascular Disease has held no meetings.

This Committee conceivably could be involved in the regional programs involving Heart Disease, Cancer and Stroke. So far this Committee has not been asked to participate in the initial planning phase of this program.

Respectfully submitted,
(Signed) ROBERT D. STORY, M.D., *Chairman*
Committee on Cardiovascular Diseases

Medical Advisory Committee to the State Highway Department

The above committee met at the Oak Manor Motel on December 11, 1966. Members attending were R. W. McLean, M.D., Chairman, and R. R. Ivers, M.D. Others present were V. J. Fischer, M.D., K. J. Kucera, State Highway Department and Myrtle Kohler, recording secretary. The breakfast meeting was convened by the Chairman, Dr. McLean.

During the past year Mr. Kucera of the State Highway Department has forwarded to the members of the committee a number of questionable cases for medical evaluation of drive-license applicants. The majority of the cases involved epilepsy or loss of consciousness. All members of the committee responded well and there was very little dissension in the committee recommendation to the Highway Department. It is felt by the committee that Association members should be most alert in recommending as to who is capable or not capable of driving a motor vehicle. It would be helpful if physicians, examining an applicant, would detail the applicant's physical or mental ability to drive a vehicle. This can be done on a medical report without making recommendation for revocation of license. A physician expressing an opinion and recommendation could be considered as libel.

Mr. Kucera stated that he had discussed some of these problems with both physicians and lawyers. In some doubtful cases it was felt that both could work together in assembling information by family interviews and impartial hearings.

Dr. Ivers suggested that some type of legislation be established in the future to protect the physician in his recommendations.

It is felt that private physicians should assume greater safety moral responsibility and become more aware of their important role in the medical aspects of traffic safety. Except in isolated cases, there has been little public awareness or interest in the medical aspects of driver licensure. Participants felt that the public attitude must be changed concerning traffic safety and its closeness to medicine.

The Chairman of this Committee is pleased to note that some accomplishments were made during the last legislative session for the improvement of safety responsibility. The neurologists and neurosurgeons were active and instrumental in some of this legislation, especially in the field of protective equipment for motor cyclists.

It was the consensus of the group that the committee should continue to function in the same manner as in the past.

Respectfully submitted,
(Signed) R. W. MCLEAN, M.D., *Chairman*
Medical Advisory Committee to the State Highway Department

Report of the Representative to the Crippled Children's Services Division Public Welfare Board of North Dakota

The Medical Advisory Council on Crippled Children's Services met May 21, 1966, in Bismarck, North Dakota. The members present were: Dr. James R. Amos, State Health Officer, Bismarck; Dr. James A. Barnes, Pediatrician, Minot; Dr. Frank D. Naegeli, Urologist, Minot; Dr. G. E. Tolstedt, General and Thoracic Surgeon, Bismarck; and Dr. Donald E. Skjei, General Practitioner, representing the State Medical Association, Williston. Dr. Malcolm Cameron, General and Thoracic Surgeon, Minot, was present by special invitation.

INFORMATION ON AND DISCUSSION OF TITLES XVIII and XIX: Discussion by Mr. Leslie O. Ovre, Executive Director, concerning Title XIX called "A Medical Assistance Program", was carried on. He states that the Public Welfare Board has been doing the same type of thing for the past twenty years. This bill includes medical and dental care and involves ¾ federal funds and ¼ state funds. It is estimated that 1½ million dollars a year will be saved in state and county funds through Medicare coverage. Medically needy children under twenty-one years of age can be included in the Medical Assistance Program. The number of medically needy children in North Dakota is an unknown figure, but could obligate the state for 1½ to 2 million dollars a year or considerably more. There was a discussion as to whether Title XIX would phase out crippled children's services. It was discussed, but the Department of Health, Education, and Welfare provides that the Crippled Children's Services' program is to be maintained and expanded. This includes maintenance of a "high quality of care" feature. It is felt that expansion of the program would be difficult in North Dakota because North Dakota already has a complete and comprehensive program. There was some discussion as to qualifications or requirements under Title XIX. Mr. Ovre stated that the only requirement is that the individual be medically indigent using an income of $1600 for one individual plus $400 for each additional individual in the family. At the present time, the Department does not know what will be done in terms of Title XIX. Conclusion of the discussion was that any changes made on the scope of the Crippled Children's Service Program would be considered by the Medical Advisory Council.

SERVICES FOR INDIVIDUALS WITH CARDIO-

VASCULAR DISEASES. This discussion was brought up in regard to the frequent referral of cardiac patients covered under Crippled Children's Services to out-of-state centers by both Board Certified and Board eligible Pediatricians and Internists because of a limited number of certified cardiologists in North Dakota. Dr. Malcolm Cameron discussed the standards of the American Heart Association, and outlined how the Minot group is meeting these requirements. In fact, Dr. Cameron felt that the cardiac care in North Dakota is better than is provided elsewhere because the service is closer to the patient's home. He stated that the Northwest Clinic anticipates eventually doing everything in terms of cardiovascular care, and does not want to do catheterizations unless the patient wishes to stay in the state for cardiac surgery. Miss Lister cited the Children's Bureau's recognition of a change in need for regional cardiac centers and that such may go in existence in time. Dr. Freise pointed out that Crippled Children's Service cannot disregard the regional heart center in economic standpoint because diagnosis in surgery in these centers is being paid from special federal funds, and does not come out of the federal money specifically granted to the North Dakota Crippled Children's Service.

PROVISION OF HOME THERAPY FOR CYSTIC FIBROSIS PATIENTS. There are presently ten children-approved Crippled Children's Service's assistance for treatment of cystic fibrosis in the state at a total yearly cost of $13,000. Dr. Freise had had a telephone conversation with Dr. Warren J. Worlick at the University of Minnesota Hospital. Relative to home therapy, Dr. Worlick informed him that equipment on therapy is available at $280, and that the use of such involves less frequent hospitalization. The effectiveness of home therapy is dependent upon the ability of the parent. Dr. Amos brought up the fact that the public health nurse could be helpful in motivating the family in following the home program. Dr. Skjei inquired if expanding the program to including home therapy for a child with cystic fibrosis would affect the total program. Dr. Freise explained that at the present time, the Crippled Children's Service Program does not include a provision for home equipment and out-patient drugs for any diagnosis. Dr. Skjei indicated a need to be selected if the program is to include home therapy, and that such be done on a temporary basis and be evaluated at the end of the year. The group concurred in the value of home therapy for the child with cystic fibrosis, and recommended that such be given further consideration by the Public Welfare Board.

SUPPORTED CARE PRIOR TO SURGERY. Dr. Freise opened this portion by pointing out some of the program problems which have been encountered in terms of supported care for hospitalization required prior to surgery such as a new-born with a cleft lip and palate condition or meningocele. After considerable discussion, the group concurred with Dr. Freise's thinking that Crippled Children's Service not get involved with the treatment other than the primary condition. Directly related illness could come within the scope of Crippled Children's Service coverage.

NURSING HOME CARE IN LIEU OF HOSPITALIZATION. Dr. Tolstedt favored expanding Crippled Children's Service to include payment for "nursing home care in lieu of hospitalization" when such is indicated.

FURTHER DISCUSSION ON OTHER MATTERS. Dr. Tolstedt favors the utilization of in-state services for cardiac patients, but indicated the need to keep in mind that every case of open-heart surgery costs between $10,000 to $20,000. He believes that there is a real problem and need for a physician and anesthesiologist if cardiac surgery is to be done, and indicated that he thought a medical-social worker is also necessary.

Respectfully submitted,
(Signed) Donald E. Skjei, M.D.,
Representative

North Dakota State Advisory Council for Hospital and Related Construction

The Committee met twice during the past year, once on the 14th of June, 1966, at which time the current and proposed hospital and old folk's home construction in the state was reviewed. The Committee decided to continue the policy of providing 46% federal support to hospitals and nursing homes contemplated for construction based on the time of the awarding of the bid for construction.

The Committee met again on December 14, 1966, and the proposed hospitals and their construction plans were reviewed.

In view of the absence of any large amount of money available at this time, no definite commitment was made. The committee did go on record as supporting the Bethel Lutheran Home for the Aged in Williston, if funds should become available during the next six months.

Respectfully submitted,
(Signed) M. A. K. Lommen, M.D., *Member*
North Dakota State Advisory Council

Report of Representative to Medical Center Advisory Council

The members of this Council are T. H. Harwood, M.D., Director of the Medical Center and Dean of the Medical School; T. L. Degman, Grand Forks attorney; James R. Amos, M.D., Bismarck State Department of Health; Mrs. V. Parker, Grand Forks, representing the North Dakota State Welfare Board; Fred R. Orth, Grand Forks, banker, representing the State Board of Higher Education; Howard Semingson, Minot, hospital administrator, representing the North Dakota State Hospital Association; Mrs. Gilman Peterson, New England, newspaper editor, representing Agriculture; and P. H. Woutat, M.D., Grand Forks, representing the State Medical Association.

STUDENTS

Of the September, 1966 class of forty-four, two withdrew. Two sophomores from the previous class failed and were dropped which is unusual in the sophomore year.

It is planned to increase the 1967 fall class to forty-seven.

Transfer of our graduates is no problem yet. There are two new two-year schools but new four-year schools will probably provide some openings for their graduates. Many of our present four-year schools have enlarged their freshman classes making fewer openings for graduates of two-year schools. There are encouraging developments from the University of Minnesota Medical School. They plan to enlarge their third and fourth year facilities and take more graduates from the University of North Dakota and South Dakota. Last year they took three students from the University of North Dakota and will take nine this year. We are advised that they hope to take twenty-five to thirty transfers from North and South Dakota schools each year at some time in the future.

President Meredith Wilson of the University of Min-

nesota has suggested that North Dakota subsidize its third and fourth year medical students at least to the extent of reducing their tuition at Minnesota to the Minnesota resident level of $550 a year. The non-resident tuition is $1200 a year.

LOANS AND SCHOLARSHIPS

The total amount loaned from Medical Center Funds since this program was instituted is $671,900. $160,961 has been repaid and only $54,000 canceled through return to small town practice. Only fifteen doctors have taken advantage of the forgiveness provision. For the 1966-1967 year $66,500 has been loaned from these funds.

Ten of our graduates returned to the state in 1966 and now about forty per cent of the physicians in the state are University of North Dakota Medical School graduates and fifty per cent of the general practitioners are North Dakota graduates. However, the record indicates that a higher percentage of our graduates returned to the state in the eight years before the loan program than in the eight years after it was started.

The AMA Loan Program has increased its interest rate to seven per cent and the maximum amount loaned reduced from $1500 to $750.

The Federal Health Professions Loan Fund has allowed us $36,000 to date. We could use $45,000 or more during the next year. These funds must be matched for ten per cent of the amount loaned. We have recommended to our loan committee that some Medical Center Loan Funds be used to match the Health Professions Loan Fund to obtain more loan money and tie up less of our funds.

The Woman's Auxiliary Loan Fund continues to increase in amount and is usually fully loaned and with a waiting list.

FACULTY

We continue to maintain a competent faculty with no more than the expected turnover. Maintaining adequate salaries is a continuing problem. We are again a little below the national average so salaries are being raised.

Ten years ago we had a faculty of twenty-three with an average salary of $9,260 and in 1966 a faculty of twenty-seven with an average of $13,900. The average increase has been about five per cent a year. The school has four Hill Professorships.

SPACE

Space is becoming a major problem. Some faculty unrest has been reported due to cramping and lack of adequate research space. Inasmuch as research space is essential to maintaining a competent faculty this is a serious problem and is under study.

There has been discussion of moving the State Health Laboratory from the Medical Center and combining it with the Bismarck Laboratory to make one single laboratory with increased facilities. While in some respects this may appear desirable, from the point of view of the Medical Center we consider it undersirable, except in that it would release space in the Medical Center for other purposes. It is our opinion that the advantages of the location of the major State Health Laboratory in close relationship with the Medical School outweigh the advantages of combining the two laboratories and that all aspects considered it would be unwise to move this laboratory from the Medical Center.

NURSING SCHOOL

Enrollment in the nursing school continues to increase. In 1963-1964 125 students were enrolled, in 1964-1965 140 and in 1965-1966 151. Further increases are anticipated. The budget for this year was $145,000 and next year will be about $180,000.

REHABILITATION CENTER

There has been a big increase in the use of the Rehabilitation Center since the arrival of Dr. Donald Barcome as Medical Director. During the past year this facility has been filled most of the time and there was usually a waiting list. The high quality of work continues and there is now a formal staff organization including essential specialist consultants and regular staff meetings.

As with all other hospitals, costs continue to rise.

The State Board of Higher Education has directed the Rehabilitation Center to make itself self-supporting on the basis of thirty-six filled beds. Rates will soon be increased in an effort to do this and it appears possible that this may be accomplished. However, it should be realized that due largely to the nature of the patients admitted to such institutions, the national average is about seventy to seventy-five per cent self-supporting.

A new administrative setup in closer correlation with the business offices of the University and with a business administrator assigned to the Rehabilition Center is resulting in better business administration and improved collections.

FINANCES

For a number of years now the cost of all Medical Center operations has been greater than the Mill Levy funds received. During the early years a healthy surplus was built up and even after using considerable amounts for construction, adequate funds were available to supplement the Mill Levy until the past year. It was predicted to the last two sessions of the Legislature in committee hearings that the time was approaching when it would be necessary to request a supplementary appropriation from the Legislature for operation of the Medical Center. This became necessary this year.

The annual budget for the 1966-1967 year is approximately $1,000,000 which requires a supplementary appropriation of over $300,000 a year which was requested from the Legislature and granted.

It should be emphasized that operation of the Medical School alone could still be done with the present Mill Levy funds. However, the Center has numerous other duties implied in the original Mill Levy Bill and others added by the State Legislature. As above mentioned, the Nursing School alone is taking $145,000 and the next year will take $180,000. Mill Levy funds also finance the Medical Technician training program and a great deal of teaching is required from the Medical School faculty for nonmedical school students. This includes various courses to Nurses, Medical Technologists, Occupational Therapists, Physical Therapists, Physical Education and Home Economics majors and others. About five hundred such students are taught various courses in the Medical School each semester.

This has all been pointed out many times in reports from the Dean, and the utilization of Medical Center Mill Levy Funds has been well documented for Legislative Committees and others. We hope that the physicians and the general public can be fully informed about the varied duties requiring time and funds from the Medical Center.

We expect that requests for increased facilities and funds for the Medical Center will continue as will those

for primary, secondary and other education and we fear that there may be no end.

Respectfully submitted,
(Signed) PHILIP H. WOUTAT, M.D.,
Representative to the Medical Center Advisory Council

Report of Representative of Blue Shield Board of Directors

As medicine is faced with changing economics and growing participation or partnership with government in the financing of medical care, so, too, is North Dakota Physicians Service.

Perhaps the greatest change this past year has been the introduction of Medicare. North Dakota Physicians Service was named the carrier and will have twelve months' experience with the program June 30, 1967.

In spite of the additional time spent on Medicare by the Blue Shield staff, to implement the many directives and changes constantly occurring, Blue Shield has had an enrollment gain of some 10,000 contracts in its regular business. It is interesting to note that approximately 40% of the population is handled by Blue Shield through its basic contracts and Medicare. The Plan ranks 16th in the United States by percent of population enrolled.

Anticipating changes in doctors' billings and increasing costs, Blue Shield's actuary recommended a change in premium effective January 1, 1967, to be made on subscribers' billing dates and groups' anniversary dates throughout the year. By request of many subscribers, coverage was included in all contracts for sons and daughters, of any age, of a family subscriber, attending school, and coverage for totally disabled sons and daughters. Recognizing that most of the reasons for the plan A contract have been eliminated by Medicare—both Title XVIII and Title XIX—plan A was merged into plan B. For matters of efficiency and to eliminate much confusion, plan C was merged into plan D. Reimbursement to doctors was based on 80% of the reasonable fee for services covered and rendered under the merged plans A and B, called Series 80. The reasoning behind the offering of a contract (Series 80) that pays doctors 80% of their reasonable fees for full service is an attempt to keep the price of medical care at a level people with family incomes less than $6,000 can themselves pay without government help. It behooves us all to develop and offer acceptable prepayment medical coverage for people with low incomes; otherwise, the demand will increase for governments to provide health care programs.

For merged Plans C and D, called Series 11, reimbursement to doctors is based on 100% of the reasonable fee.

This change in method of payment from a rigid fee schedule to the method applied by law to Medicare will have a gradual tendency to lessen paper work and different billings for different programs.

The North Dakota Legislature met in biennial session this year and North Dakota Physicians Service was faced with a greater number of introduced Bills than at past sessions that would have affected the program, those most important (all defeated) being:

1. S.B. 151—Recognizing and paying Podiatrists for covered services furnished Blue Shield subscribers.

2. S.B. 227—Requiring the Welfare Department to contract with an outside agency for administration of Title XIX. (The Welfare Department intends to administer Title XIX themselves.)

3. H.B. 586—Recognizing and paying optometrists for covered services furnished Blue Shield subscribers.

4. H.B. 680—Osteopathic

5. H.B. 872—Giving to the Insurance Commissioner power to regulate premium rates and, indirectly, doctors' fees.

6. Senate Concurrent Resolution 000—Directing the Legislative Research Committee to make a study of whether Blue Shield should be compelled to recognize and pay paramedical people.

As hearings were held on these Bills, one gathered that this is not a diminishing area of concern but one requiring greater watchfulness. It is felt by the Blue Shield Board of Directors that a full-time lobbyist should be obtained by Blue Shield to represent the corporation throughout the next session. It might be pointed out that North Dakota Physicians Service did obtain the services of a full-time lobbyist after the present session had begun, who did a very satisfactory job.

Some criticism was leveled at North Dakota Blue Shield for the reason that the Board of Directors is composed of 100% physicians. Recognizing this as a possible future problem, an Ad Hoc Committee has been formed from members of the Blue Shield Board to report on this matter at the Annual Corporate Meeting of Blue Shield.

Perhaps the most pressing problem that the Plan has faced has been determining reasonable fees as defined and interpreted in the Medicare Law. Briefly, the law states that two criteria must be considered in determining reasonable charges:

1. The customary charges for similar services generally made by physicians,
and

2. The prevailing charges in the locality for similar services.

The law also specifies that the reasonable charge cannot be higher than the charge applicable for the carrier's own policyholders for comparable services under comparable circumstances.

A great deal of confusion has developed as to a determination of "reasonable" fees by Blue Shield as it applies to the basic Blue Shield subscribers' contracts and to Government Medicare Part B. In order to clarify the subject, the Blue Shield Board of Directors approved the following guidelines:

I. Since Blue Shield has never paid for office calls in any of its basic subscriber contracts either before or after surgery, the "reasonable" fee determination for surgery will be on the basis of "prevailing" fees to include the surgery and up to two weeks necessary after-care in the hospital. No allowance will be made for anticipated follow-up office calls. It would be the doctor's privilege to bill the patient for these calls and they would be paid for by the patient to him.
Should the patient require hospitalization beyond the normal two weeks post-operative period, medical calls for this additional care should be itemized on the claim and consideration for additional payment will be made by Blue Shield.

II. Government Medicare Part B does allow payment for necessary office calls but does not allow "prepayment" for office calls anticipated in the future. Payment for surgery would be on the basis as outlined above. In addition, payment of the "reasonable" fee for necessary office visits following surgery

will be made but such visits must be itemized and billed to either Medicare Part B or to the patient.

Respectfully submitted,
(Signed) Phillip O. Dahl, M.D.
Representative of the House of Delegates to the Board of Directors of Blue Shield

Committee on AMA-ERF

No formal meeting of this Committee was held during the past year of 1966-67. The medical school at the University of Grand Forks receives, at various times during the year, funds which have been designated by Alumni and donations from North Dakota practicing physicians. Also received are undesignated funds from the AMA Medical Education Department. In 1963, this was a total of $6,000; 1964—$4,000; 1965—$4,283; and in 1966—$3,361 designated and $1,426.76 undesignated. In examining these figures more closely, it would appear that the last three years have shown a decrease in funds received. During the past year, $403 was received in July, 1966, and $334 in March, 1967.

We have no figures to present on a national scale; but as we have not received any notification from the AMA office, we assume this decline is not national.

At this time, we would like to make an earnest plea to the doctors to become more aware of their obligation to medical education. Any gifts sent in and designated for the University Medical School will be transferred immediately. We wish to thank the North Dakota doctors in advance for their contributions.

Respectfully submitted,
(Signed) C. M. Lund, M.D., *Chairman*
Committee on AMA-ERF

Committee on Medicine and Religion

During the past year there has been considerable activity in the cities and counties as most hospitals now have a working relationship between the man of medicine and the man of religion. Several of the hospitals, by the action of the doctors, have a full or part time chaplain.

It is sincerely hoped that all doctors will endeavor to continue the good relationship now existing with the ministerial associations in our state.

It was my pleasure to attend the conference of all the 50 state chairmen, February 18-19 in Chicago. At that time, representatives from all denominations, Bishops, Rabbis and Ministers were also in attendance. It was agreed upon by all, that the patient be treated medically and spiritually by the physician in each category.

There are plans to furnish each physician with a booklet containing a brief outline of the various religions with pertinent facts relevant to the physicians management of his patients.

During the past legislative session, there were several bills that were of considerable interest to this committee. However, due to past policy it was decided that this committee should not make a decision on subjects with possible political implications. At the meeting of the State Chairmen in Chicago, we were asked to be active politically to insure the welfare of our patients medically and religiously.

It is of interest that the present AMA president-elect, Milford O. Rouse, M.D., was the founder of this committee, and was in attendance at our meeting in Chicago. Dr. Rouse, speaker at our banquet, asked us to help him in getting all physicians to treat the "total patient." All doctors are invited to read a series of ten articles on Medicine and Religion beginning in the April issue of JAMA.

Respectfully submitted,
(Signed) G. H. Hilts, M.D., *Chairman*
Committee on Medicine and Religion

Resolution from Stutsman County Medical Society

The following resolution has been passed by the Stutsman County Medical Society:

The Society recommends to the State Medical Association that the Legislative Committee meet at appropriate times ahead of the interim meeting in the legislative year so that the legislative program can be well formulated, the members of the House of Delegates and the Council be fully informed of it prior to the interim meeting and that known legislation proposed by other healing professions could be considered and methods for handling it be formulated before the legislators actually convene. This information could be forwarded to the constituent County Medical Societies and each Society should take appropriate action.

COMMITTEE REPORT
Ad Hoc Committee for Preparation of Application Heart Disease, Cancer and Stroke Program

This Committee was appointed by President Glenn W. Toomey in November of 1966. The first meeting of the Committee was held in Fargo, North Dakota, on December 9, 1966, at which time during a four hour meeting, the members of the Committee were all oriented as to the nature of the legislation for the Heart Disease, Cancer and Stroke Program and various opinions of the members were polled as to the course of action which should be taken with respect to the preparation of the application.

It was moved, seconded, and passed that Doctor T. H. Harwood be nominated as Program Coordinator for the Heart Disease, Cancer and Stroke Program.

It was moved, seconded and passed that the University of North Dakota be appointed as Fiscal Agent for the Heart Disease, Cancer and Stroke Program and preliminary plans for the formulation of the application were developed.

The action of the Committee was then presented before the Council on December 10, 1966. At this time the Council allocated funds for the use of the Committee in preparation of the application and approved the selection of the University of North Dakota as Fiscal Agent and gave the Committee the prerogative of election of the Program Coordinator. Various members of the Committee were then assigned portions of the application and as various portions of the application were completed, the membership of the Committee was circularized by mail for perusal and approval.

A sub-committee was developed and a conference call of the sub-committee was held in order to develop The Plan, The Budget, and The Relationship of The Budget to The Plan. After these portions of the application had been developed, the preliminary draft of the application was printed and bound and a second meeting of the Committee was held in Bismarck, North Dakota, on January 29, 1967.

At this time, the various sections of the application were carefully perused, minutely corrected and approved. Additional sections of the application were assigned to Doctors A. Malcolm Cameron, J. D. Craven and T. H.

Harwood. After these sections had been received and the corrections made as per the recommendation of the Committee as a whole, a meeting was called of the Regional Advisory Group and all members of the Ad Hoc Committee on February 19, 1967, in Fargo, North Dakota.

At this time, the booklet had been prepared in semi-final form and bound, and each member present at the meeting was given a copy of the book. Section by section, page by page, the book was reviewed, corrected, revised and approved. After this final perusal and revision the motion was made by Mr. Thomas L. Degman, member of the Advisory Group, and seconded by Sister Mary Adrian, member of the Advisory Group, and unanimously passed that the application book in its revised form be prepared and submitted for a Planning Grant amounting to a total of $386,469 over a two year study period. The amount requested for the first 12 months period was $188,010. In keeping with the motion, the Chairman made the necessary revisions.

Dr. A. Malcolm Cameron produced some excellent printed maps showing the North Dakota regional and sub-regional hospital towns, as well as the North Dakota referral area. Dr. T. H. Harwood completed the section on The Program and Progress Report Preparations. The 66-page book was printed by an off-set printing method and a total of 75 copies were made. The original and four additional copies were sent to the National Institutes of Health, and the remaining copies were sent to all members of the Board of Governors of the North Dakota Medical Research Foundation, all members of the Ad Hoc Committee, all members of the Advisory Group, as well as the President of the North Dakota State Medical Association and the Speaker of the House of Delegates of the North Dakota State Medical Association.

The total cost of the preparation of this application was approximately $1500, which included the travel fare and three days of per diem reimbursement at $9 per day to the Chairman of the Committee, these expenses being incurred as a result of his attendance at the National Conference on Regional Medical Programs held in Washington, D.C., January 13-17, 1967, and the expenses being authorized by unanimous vote of the Ad Hoc Committee.

As Chairman of this Committee, I wish to commend all members of the Committee for their cooperation, diligence and extended efforts in the preparation of this application and it is the sincere hope of the entire Committee that the application will meet with the approval of the State Association and will be looked on favorably by the National Institutes of Health. It is also the sincere hope of the Committee that if the application for planning funds is successful, these funds will be utilized in a manner that will prove truly beneficial for our North Dakota region.

Respectfully submitted,
(Signed) Lee A. Christoferson, M.D., *Chairman*
Ad Hoc Committee on Heart Disease,
Cancer and Stroke

REPORT OF THE DELEGATE TO THE AMERICAN MEDICAL ASSOCIATION

The AMA House of Delegates met in June and November. The volume of resolutions and reports and the depth they were considered is belied by the necessary brevity of this report; however, they were adequately covered in JAMA and AMA News. I would like to separate this report and first list recent actions of the AMA House which are of interest to State and District Medical Societies. I shall furnish the reference committee chairman with detailed information on each subject for his committee's consideration and action.

1. Measles immunization
2. Non-physician lab directors—Physician leadership in blood banking
3. V. D. control
4. Exercise and physical fitness programs
5. Medical Society review committees
6. Utilization committees—function, immunity, etc.
7. Title XIX—direct billing, free choice, et al.
8. Chiropractic—an unscientific cult—Quackery conferences
9. Explorer Scout Program oriented towards medicine
10. Health manpower
11. Hospital planning—long range—local and state
12. Alcoholism—treatment in General Hospitals
13. Nurse salary increases—support for
14. Physician membership on hospital governing boards
15. Local societies response to unfavorable public image—news media
16. Onus of federal health legislation failures on federal planners, et al.
17. AMA Voluntary Physicians to Vietnam Program
18. Utilization of retired physicians and nurses
19. Medical Society Review Committees
20. Regional Complexes
21. Elderly applicants for auto liability insurance—determination of
22. Millis Report (Committee on Medical Education)
23. Survey Drug costs
24. Emergency Medical Care
25. Individual Responsibility—statement of
26. Official public commitments to better health care (Doctor C. Hudson)
27. AMA-ERF
28. Abortion laws

The AMA House of Delegates meets under threatening sociologic clouds of lay schemes and proposals to make health care more readily available to every person and to administer the public health care dollar more efficiently. Medicare remains but a financing mechanism; it does not provide services. There have been some rough spots in its implementation, particularly in matters of direct billing, utilization review, certification and recertification. The House is concerned about the vexations of Medicare, but more concerned about its expansion into a national health system. Much will be said about the increasing cost of health care, and we physicians face a complex task in trying to influence the cost of health care. On the one hand, we are asked to use our influence to gain for nurses better wages; on the other hand, we are asked to moderate drug costs to our patients. Nevertheless, no one is in a better position to exercise control over the cost of health care than the physician. The contention of the medical profession that it can police itself is under severe and direct challenge on Capitol Hill in the Senator Hart Hearings. Rather profound implications are seen should this bill prevail. Repeatedly, the AMA has stated that the interest of the patient comes before the interest of anyone who serves him. The House recognized the need to increase the number of physicians who "treat patients", and proposed specialty status for the family practitioner together with new training programs. Medical education will be soundly thrashed out in June as the Component Medical So-

cieties report on the Millis Report (under consideration by our Committee on Medical Education).

It's been rumored that the AMA Disability Insurance Program is "up for grabs." I'd be less than honest if I admitted more knowledge of this than any of you. I doubt that any decision will be made before June, and I so requested of the Board of Trustees, but I must also admit to being in sympathy with their action.

What is the thinking of the AMA House? The only sure thing at this moment is that affairs will not be tomorrow as they are today. Death and taxes used to be the only two certainties, now there is a third, change. The AMA House recognizes the inevitability of "change" and the necessity to steer and guide it constructively. Success will be completely dependent upon the strength, dedication and commitment of component medical societies to better their health care services.

My thanks to Doctors Bob Painter, Jim Moses, Leonard Larson, Willard Wright, Glenn Toomey and to Lyle Limond for helpful coverage at these meetings.

Respectfully submitted,
(Signed) Thomas E. Pederson, M.D.
Delegate to the American
Medical Association

Committee on Emergency Medical Service

Your Chairman attended the A.M.A. Conference on Emergency Medical Service in Chicago on April 6 and 7, 1967.

This was primarily a planning conference on future emergency service. A condensed version of the meeting will appear shortly in the Journal of the A.M.A.

Respectfully submitted,
(Signed) Perry H. Engstrom, M.D., *Chairman*
Committee on Emergency Medical
Service

Committee on Medical Economics

The Medical Economics Committee of the North Dakota State Medical Association met on three occasions in 1966 at the Prince Hotel, Bismarck, North Dakota. The dates of these meetings were as follows: 3/19/66; 6/18/66; and 9/17/66.

On all three occasions Mr. Donald Eagles, Executive Vice-President of the North Dakota Physicians Service, presented comments and answered questions regarding Blue Shield and Blue Shield's role as Carrier for Medicare, Part B. Mr. Leslie O. Ovre, Executive Director, Public Welfare Board of North Dakota, and Mr. Gottfried J. Kuhn, Director of Medical Services, Public Welfare Board of North Dakota, were present at the meetings of 6/18/66 and 9/17/66 and answered questions from the committee regarding Title 18 and Title 19 of Public Law 89-97.

At the meeting of 3/19/66 the committee endorsed the Confidential Survey of Usual and Customary Fees sent out by Blue Shield to assist them in developing a "profile" of charges made by physicians for use in Blue Shield's role as Carrier for Medicare, Part B. At that same meeting a group of anesthesiologists appeared and presented their views on anesthesia values based on time units only.

At the meeting of 9/17/66 representatives from the Vocational Rehabilitation Department presented their arguments and need for a definite conversion factor in order to satisfy their auditors. Also at that meeting Mr. Robert Bain of the Byrne Insurance Company of Bismarck, North Dakota, presented a plan of accidental death and dismemberment insurance to be made available to the members of the North Dakota State Medical Association on a voluntary group policy basis. The committee approved participation in the program with the final approval to be obtained from the Executive Committee of the Council. The Committee went on record disapproving the action of the North Dakota Public Welfare Board at its August, 1966, meeting in which they granted modest increases in conversion factors for the Crippled Children's Services program.

Also at the meeting on 9/17/66 the proposed modifications to the 1964 California Relative Value Studies, which had been studied for months, were approved. Four changes in the unit values were made in the Medical Section. A new section on Anesthesia was developed, based on time units. In the section on Surgery the column dealing with anesthesia was deleted as were footnotes relating to calculations of total anesthesia value. Twenty-nine procedures were changed in unit value in the Surgical Section. Minimal changes in terminology were also made in this section. The title "Laboratory" was changed to read "Clinical Pathology and Pathologic Anatomy". The title of the book was changed to Relative Value Guide instead of Relative Value Schedule for reasons noted in the foreword of the book. (The October, 1966, edition of the Relative Value Guide was published and then distributed to all doctors on 11/4/66).

On 9/21/66, one of us, (O. V. Lindelow, M.D.) met with the Public Welfare Board at the Capitol Building in Bismarck. The very strong feelings of the Medical Economics Committee and the doctors in North Dakota in regard to the philosophy of the usual, customary, and reasonable fees for services given to welfare clients was again emphasized to the Board. On 9/22/66 we were informed by Mr. Leslie Ovre that the Public Welfare Board had agreed to the use of usual, customary, and reasonable fees commencing on 10/1/66 and that they would accept the Relative Value Guide to be published in October, 1966. All doctors in North Dakota were informed by news letter from the Executive Secretary of this change in payment by the Public Welfare Board. This agreement does not apply to the Crippled Children's Service since this is a separate program and specialist participation in the program is voluntary, and conversion factors have never been officially negotiated with the Crippled Children's Service. The conversion factors in the past have been uni-laterally developed by them.

Vocational Rehabilitation is paying usual and customary fees and using the 1966 Relative Value Guide. It is understood that the Indian Bureau, Workmen's Compensation Bureau, and Veterans' Administration are now paying usual and customary fees.

Blue Shield is now paying usual and customary fees, basing their payments on the "profiles" of the doctors' charges developed from their confidential survey of usual and customary fees. This is the "profile" that is also being used for payment of claims for Medicare, Part B. Since 1/1/67 Blue Shield has had two plans instead of four. The lesser plan pays 80% of the usual and customary fee. On 3/18/67 the Blue Shield Board of Directors approved payment of a technical surgical assistance fee. Payment will be made on the basis of not to exceed a maximum of 15% of the reasonable fee of the physician in charge of the case.

On 4/3/67 a meeting was held with General Norman E. Peatfield, Executive Director of ODMC, for discussion of the schedule of maximum allowances. Those in attendance at the meeting in Bismarck, 4/3/67, included: General Norman E. Peatfield, Col. Robert Holzworth,

Mr. Lyle A. Limond, and O. V. Lindelow, M.D.

The state office of the North Dakota Medical Association had previously mailed out a Fact Sheet on the new Uniformed services Health Benefits Program. This Fact Sheet outlined the health benefits authorized by Public Law 89-614 (Military Medical Benefits Amendments of 1966) for the dependents of active duty members of the Uniformed Services, for retired members, and the dependents of retired and deceased members of the Uniformed Services.

The Office For Dependents Medical Care (ODMC) now has a new title in view of the new program. This Title is Office for The Civilian Health and Medical Program of the Uniformed Services (CHAMPUS).

On 12/21/66 Mr. Lyle A. Limond mailed General Norman E. Peatfield a copy of the Statement of Policy adopted by the Council and the House of Delegates of the North Dakota State Medical Association on Saturday, December 10, 1966, and Sunday, December 11, 1966, regarding the ODMC contract. This statement of policy emphasized that it had been ten years since the initial Medicare fee schedule was negotiated and that the economic picture had changed. This statement also emphasized that our position regarding fees had not changed, that is that doctors should bill and be paid their usual and customary reasonable charges. It was also emphasized that the 1966 Relative Value Guide should be used in negotiating the new contract.

On 1/30/67 an extension to the old contract was signed and forwarded to the Wisconsin office. This extension will last through April 30, 1967. This was signed so that there would be time to meet with CHAMPUS officials so that agreement on a new schedule of maximum allowances could be made.

In answer to a question, General Peatfield stated that the new Uniformed Services Health Benefits Program would cover from 6 to 6½ million people but that perhaps 50 to 60% of these would be active participants or approximately 3 million people.

General Peatfield stated that CHAMPUS would accept the October, 1966, Relative Value Guide of the North Dakota Medical Association as far as coding and procedural names with some necessary additions that they must make to this guide.

A maximum conversion factor was discussed to help CHAMPUS develop a maximum schedule of allowances for North Dakota. By law they must have some maximum dollar figure for procedures and services. This is similar to the needs of Vocational Rehabilitation office. An agreement was made on a maximum conversion factor. This is confidential and will be unpublished. There are obvious reasons for not letting the figure be general knowledge. The schedule of maximum allowances as will be put out by CHAMPUS will contain actual dollar maximums.

CHAMPUS will also need some guide to determine reasonableness of fees in a particular situation. Basically what is needed is a profile of fees for individual doctors such as is now used by Blue Shield and Medicare Part B which is run by Blue Shield in North Dakota. It was pointed out that in the confidential survey of usual and customary fees made throughout the nation one year ago that it was found that most fees were increased by about 20% or so from their previous levels. This is one of the faults of such a survey, particularly when dealing with the government in our present economic and political situation. Reasons for this were discussed.

The confidential survey of usual and cutomary fees carried out by Blue Shield in March, 1966, is of course confidential and cannot be obtained by CHAMPUS. However, since such a profile of fees is a requirement it was felt that the Medical Economics Committee and the Executive Secretary of the North Dakota State Medical Association could perhaps contact Blue Shield and obtain material from this previous survey to be used in evaluating submitted charges for the program of CHAMPUS. Blue Shield will be contacted in this regard.

In further discussion it was pointed out that a particular doctor's profile of fees can be changed. This has been done in other ODMC programs throughout the nation. It is done by the individual doctor giving 90 days notice to the third party involved, that his fees will go up to a certain level and stating this level.

Most of the experience in North Dakota with the previous ODMC has been in orthopedic work and obstetrics. Thus one could not develop a profile of fees of individual doctors in regard to internal medicine from this previous data. This is why a profile of fees for individual doctors must be obtained from other surveys.

The cost of injectable medications given by the doctor in his office was discussed. Evidently it has been the custom in the past that ODMC would pay for only the cost of the medication and not for the technical or professional charge of administering such a drug. In the past it has apparently been the custom, mainly by older practitioners, to include the cost of the injectable medications in the office fee. General Peatfield was informed that this is not the custom in this area and that injectable medication is charged separately from the charge for an office visit. This was new to him, but he promised to investigate it and evaluate the possibility of paying for injectable medication separately on a usual, customary, and reasonable basis rather than just paying for the cost of the medication. The reasons why he should do this were discussed.

It should be pointed out that under the new CHAMPUS program medications will be paid for providing they are prescription medications and obtained from the pharmacies. The only medication that will be paid for that does not require a prescription will be insulin, for obvious reasons. If a dependent is ordered overseas, or has orders for going overseas, the cost of immunization shots is payable under CHAMPUS. These shots are evidently paid for on the basis of usual and customary fee. It is hard to correlate this practice with the previous practice of paying only for the cost of medication and not for any technical or professional component of giving an injectable in the office.

General Peatfield stated that he would develop the new contract embodying the ideas that were discussed at this meeting. An attempt will be made to obtain a profile of fees to be used by Mr. Lyle A. Limond in administering the CHAMPUS program for North Dakota.

A Claims Review Committee will have to be set up for CHAMPUS. Possibly this could be tied in with the Claims Review Committee now functioning for Blue Shield's Medicare Part B.

Glenn W. Toomey, M.D., President of the North Dakota State Medical Association has appointed a Medical Advisory Committee to the Public Welfare Board on Title 19. This committee consists of 12 members, 6 of whom are members of the Medical Economics Committee.

Respectfully submitted,
(Signed) O. V. Lindelow, M.D., *Chairman*
Committee on Medical Economics
and
James F. Houghton, *Vice-Chairman*
Committee on Medical Economics

Committee on Legislation

This Committee has been fairly active this year although they had only one meeting during the Legislative Session. There has been some activity both on a local and a national basis by individual members of the North Dakota State Medical Association and there has been a good deal more interest taken by the membership at large in various medical legislation than in the past. I did contact our representation in Washington on several issues and all in all I received a comparatively good response. I do, however, feel that some of our representation needs a little bit more education insofar as various methods of approach are concerned.

I am still concerned by socialization of medicine which I had given in my 1964 report indicating that at that time there was definitely more objection by the Medical Association, both state and the American Medical Association relative to the support of the aged segment by Social Security medical legislation. This has now become a reality. At the very present time before Congress of the United States in committee and being evaluated by this committee are various amendments to the Social Security approach, or Medicare. These consist primarily of lowering the age, which is at the present time 65, and also for the purpose of taking care of the segment which has Social Security disability. This would open the wedge just a wee bit wider than it has been in the past, and this would include all individuals immaterial of age. There is also in this same bill another segment which would give Medicare assistance to all widows and all children up to the age of 21. Needless to say, this will open up a considerable segment of our population. It appears that one were cutting off the dog's tail one inch at a time because there would be less pain connected with this. In the same breath, there are several things in the offing which would be in accord with the medical boys, such as the doing away with recertification of Medicare patients who are in the hospital. I do feel that there has been a gross expenditure of tax money of the people in the State of North Dakota by those who process a number of these claims by telephone—I presume to expedite matters. However, this does cost money and with the numbers of practitioners in the state, would be repeated many times per day throughout the entire state, necessarily increasing the expense involved. I shall not go into detail as to whether this meets with my particular approval or not.

I would indicate to most of the membership that the recent legislative session was not a total success as far as medical legislation was concerned. Bill Buckingham did a very noble job and frankly should have his own report in here because he was much closer to the scene of battle than I was, and could undoubtedly give a much better account of the running battle than I could. There were many things observed, many things supported and many things that were objected to by the Medical Association at the recent Legislative Session. Accompanying this report will be the bills indicating what action was finally taken insofar as these are concerned.

One of the things that went by the wayside was our legislation regarding abortion law changes in the State of North Dakota. I would say that on a National basis, therapeutic abortion laws present considerable confusion. I have a good deal of material regarding this, and I presume all of these factors will be taken up by the commission which was appointed to study this for the next two years.

I have before me a report from Dr. Lee A. Christoferson, Speaker of the House 79th Annual Meeting, North Dakota State Medical Association, April 21, 1966, where he has come to the conclusion as far as his committee, that teaching standard medical principles and practices, osteopathic, in this country in theory corresponds roughly to that of physical medicine. This is being subjected to scientific evaluation. There are, however, limitations of physical facilities and the clinical teaching material in most of these osteopathic institutions and as a result the recommendation of the committee is contrary to any concept of setting up a joint board in the State of N. Dak. The conclusions of Dr. Nuessle and Dr. Cleary were that they believed that if equal practice privileges were granted to both M.D.'s and osteopaths there should be a requirement of equal standards for licensure. They found that of the five osteopathic schools in the United States that were studied, they did not meet the minimum requirements for medical schools. The establishment of a composite examining board would be giving approval by the North Dakota Medical Association of osteopathic schools as equal in training and standards when they are truly not so. They believe it would be prejudicial to the interest of the people of North Dakota and to the North Dakota State Medical Association to approve a composite board in the State of North Dakota. In the last session, the osteopaths attempted again to obtain complete surgical privileges. This was beaten down again. This may happen again at every session for the next ten years and it may be beaten down, but it may also finally be approved. This would correspond in essence to the total socialization of medicine which was propounded many years ago and fell by the wayside but which had 21 points of socialization. We now find after approximately twenty years that of the 21 points 14 points have been consummated and are now a matter of record.

This constitutes about all I have to say except that I do feel Dr. Buckingham should have his own private observation along with this report.

Respectfully submitted,
(Signed) O. W. Johnson, M.D., *Chairman*
Committee on Legislation
W. M. Buckingham, M.D., *Vice-Chairman*
Committee on Legislation

SENATE BILLS

Number	
8	Appropriates $9,506,029 for State Hospital. Followed with interest. Governor signed.
18	Appropriates $2,017,534 for Vocational Rehabilitation Division. Followed with interest. Governor signed.
21	Appropriates $100,000 for Children's Psychiatric Clinic. Followed with interest. Governor signed.
35	Construction of a Public Health Lahoratory on the Capitol grounds. Association favored a laboratory at UND in Grand Forks. Indefinitely postponed in the Senate.
36	Transfers regulation and licensing of certain sanitary functions from the State Laboratories Department to the State Department of Health. Supported. Indefinitely postponed in the Senate.
44	Provides for suspension of drivers' license for certain types of traffic violations, classifies authority of commissioner over out-of-state revocations and cancellations; authorizes blood tests on traffic fatalities and amends code sections relating to operators licensing and appointment of agents to issue drivers licenses. Followed with interest. Governor signed.
52	Provides for compulsory testing for PKU. Followed with interest and token support given. Governor signed.
84	Classifies LSD as a restricted, dangerous drug and limits distribution to prescription by physician and limits refilling of prescriptions. Opposed. Indefinitely postponed in the House.
113	Empowers licensed physicians to authorize admission of dangerous, mentally ill persons to a private hospital or to the State Hospital when county judge or county mental health board member is not available. Followed with interest. Governor signed.
114	Repeals law making attempted suicide a crime. Followed with interest. Governor signed.

118 Appropriates $1,200,000 for child and adolescent treatment center for mentally ill children. Supported. Indefinitely postponed in the Senate.
151 Permits chiropodists to participate in non-profit medical service corporations (Blue Shield). Opposed. Killed in the Senate.
157 Establishes a State Air Pollution Control Advisory Council. Supported. Killed in the House.
190 Licensing and regulation of psychologists. Supported with amendments. Governor signed.
227 Relates to contract of the Public Welfare Board with private agencies or carriers. Association had this bill introduced. Indefinitely postponed in the Senate.
239 Relates to non-discrimination and freedom of choice of ocular practitioner. Token opposition. Governor signed.
390 Establishment of sheltered workshops. Followed with interest. Governor signed.
394 Provides for certification of dealers and persons engaged in fitting hearing aids. Followed with interest. Indefinitely postponed in the Senate.

SENATE CONCURRENT RESOLUTIONS

KKK A concurrent resolution directing the legislative research committee to study the miscarriage and abortion statutes of the state to determine whether or not they are in need of amendment. Association had this resolution introduced instead of a bill. Supported. Indefinitely postponed in the Senate.
OOO A concurrent resolution directing the legislative research committee to study the inclusion of medical disciplines under insurance contracts. Opposed. Indefinitely postponed in the Senate.

HOUSE BILLS

Number
511 Appropriates $13,918,800 for Public Welfare Board. Followed with interest. Governor signed.
518 Appropriates $68,000 for scholarships for nurses. Followed with interest. Governor signed.
525 Appropriates $897,438 for Public Health Department and $402,667 for financial assistance in establishing and maintaining mental health and retardation units. Followed with interest. Governor signed.
534 Amends bulk of laws pertaining to mentally deficient persons and care and treatment provided them. Followed with interest. Governor signed.
536 Provides for appropriation of $100,000 for purpose of initiating and planning construction of a facility for the mentally deficient. Followed with interest. Indefinitely postponed in the House.
538 Provides for Superintendent at Grafton State School to recommend a sterilization operation for a resident of such school upon receiving approval of a hoard of professional personnel and written consent of a relative or guardian. Supported. Governor signed.
549 Provides for licensing and regulation of medical laboratories and prescribes standards that directors and supervisors of these facilities must meet. Opposed. Indefinitely postponed in the House.
586 Permits insured under accident and sickness insurance to choose his own physician, surgeon or optometrist. Followed with interest. Indefinitely postponed in the House.
595 Requires protective helmets to be worn by operators and passengers on motorcycles. Supported. Governor signed.
624 Provides for formation of county-city health districts. Followed with interest. Governor signed.
680 Osteopaths given full surgical and drug privileges. Opposed. Killed in House.
691 Relates to the establishment of mental health and retardation service units in county that does not have a city with a population of 5,000 or more. Followed with interest. Governor signed.
725 Defines chronic alcoholic as a mentally ill individual. Followed with interest. Indefinitely postponed in the House.
822 Authorizes control, prevention and abatement of pollution of surface waters. Supported. Governor signed.
872 Provides that rate changes of medical service contracts (Blue Shield) must be approved by the insurance commissioner. Opposed. Indefinitely postponed in the House.
892 Residential rehabilitation facility for alcoholism at Fargo. Followed with interest. Indefinitely postponed in the House.
899 Sets standards to qualify for medical assistance care and services. Association had this bill introduced. Supported. Indefinitely postponed in the House.
902 Provides for day care centers for mentally deficient children. Followed with interest. Indefinitely postponed in the House.

Dr. Dahl: Those alternates who have been seated as delegates will serve on the reference committees on which the delegates were serving. For example: Dr. Wakefield will serve on reference committee No. 4 in place of Dr. Little. However, Dr. Lommen, Vice-Chairman of this Committee will assume the chairmanship, and Dr. Poindexter will serve as Vice-Chairman of this committee. Dr. Goltz, alternate for Dr. Jaehning, will serve on reference committee No. 1; Dr. Clayburgh and Dr. Huntley on reference committee No. 4, and Dr. Nelson on reference committee No. 5. It was suggested that the committee hearings be held on Saturday morning at 9:30 to 10:30 and Saturday afternoon from 4:30 to 6:00. It is entirely up to the discretion of the Chairmen of these committees and if they have times other than these, please mention the time now. I will appreciate it very much if the chairmen of the various committees make a special effort to contact individuals who are knowledgeable in the areas on reports they are covering so that we may get as broad a view as possible. When the reports come back for action to the House of Delegates on Sunday, then we may act with as much wisdom as we are capable of.

Last year, Dr. Foster, who is Chairman of the Committee on Constitution and By-Laws for the state was requested to bring forth some suggestions for changes in the by-laws. This committee has met and Dr. Foster has presented a resolution, which I believe each of you have. This is referred to Committee No. 6, the Resolutions and New Business Committee.

There is another resolution on the eligibility for 50 year Honors, and unless there would be an objection from the floor, and I believe you have this resolution in front of you, this would be referred to Dr. Vinje's committee No. 6.

Resolution from the First District Medical Society, which I believe you have in front of you, if there is no objection from the floor, will be referred for discussion and action to Reference Committee No. 3. Hearing no objection, it is so ordered. On the resolution on motor vehicles, which will have to be threshed out, the chair will refer this to committee No. 6. Hearing no objection, it is so ordered. The resolution from the Sixth District Medical Society, which you have in front of you, will be referred to committee No. 6. Hearing no objection, it is so ordered.

In your Handbook on page 150, you will note a resolution. I do not wish to push any needles in, but unfortunately, it is the only resolution received in time to be placed in the Handbook. This came from the Stutsman County District Society. Unless there be objections from the floor, the chair will refer this to committee No. 6.

At the Council meeting last night, our delegate to the American Medical Association, Dr. Pederson, requested that the House of Delegates take action on a resolution regarding Section 1801, Public Law 89-97. This is requesting that state associations and district societies do not allow any federal officers to supervise any control over any phase of medicine in any manner. A resolution will have to be written and acted upon by our House of Delegates. This will be referred to the Resolutions Committee, Dr. Vinje, Chairman, unless there is an objection from the floor. Hearing none, it is so ordered.

The President-Elect, whom you have just heard, has suggested two areas that he would like to see fully investigated and something effected during his year of office, such as an endeavor for long-range hospital planning in the state on a local level. We have nothing written on this, but since our President has brought this up, I am going to refer this to Dr. Vinje's committee No. 6. I have talked to Dr. Fischer and he said he would be very happy to appear before this committee and help

them to take any action they see fit. Dr. Fischer will provide the committee with some written material. Hearing no objection from the floor, this will be referred to the Resolutions Committee.

The matter of a medical society review committee was discussed at length at the Council Meeting. All of the councillors have some views on this and Dr. Fischer is particularly interested. I will refer this to Resolutions Committee No. 6. It is unfortunate that so much has to go to the resolutions committee. However, all the rest of the committees have just as much to handle. Perhaps this can be worked out in the future. Dr. Fischer said he would like to appear before this reference committee on the review committee, and also Dr. Strinden would like to appear before this committee and there are other members of the Council who are interested.

The Chair at this time will recognize Dr. Miles.

Dr. Miles: The delegates from the Seventh District have been instructed to introduce a resolution regarding the Abortion Law. They felt in view of the success in other states, that it might be well to introduce it at this time.

RESOLUTION

WHEREAS, the existing code regarding termination of pregnancy is unduly restrictive, and

WHEREAS, certain instances of pregnancy should be terminated which do not threaten the life of the mother such as pregnancy resulting from rape or incest,

THEREFORE, the North Dakota State Medical Association recommends that Section 12-25-01* be amended to read:

"A licensed physician is justified in terminating a pregnancy if:

(a) He believes that there is substantial risk that continuance of the pregnancy would gravely impair the physical or mental health of the mother or that the child would be born with grave physical or mental defect, or the pregnancy resulted from rape . . . or from incest; and

(b) Two physicians, one of whom may be the person performing the abortion, have certified in writing their belief in the justifying circumstances, and have filed such certificates prior to the abortion in the licensed hospital where it was to be performed, or in such other place as may he designated by law."

*12-25-01. Procuring an abortion – Punishment – Every person who administers to any pregnant woman, or who prescribes for any such woman, or who advises or procures any such woman to take, any medicine, drug, or substance, or uses or employs, or procures or advises the use, of any instrument or other means whatever, with intent thereby to procure the miscarriage of such woman, unless the same is necessary to preserve her life, shall be punished by imprisonment in the penitentiary for not less than one year nor more than three years, or in a county jail for not more than one year.

I move that the House of Delegates accept this resolution to be referred to a committee for action.

Dr. Hieb: I second the motion.

Speaker Dahl: All in favor? All ayes. This resolution is now on the floor and unless there is an objection, the chair will refer this to Reference Committee No. 3. Hearing no objection, it is so ordered.

You have another resolution in front of you from the First District Medical Society regarding Crippled Children. If there is no objection from the floor, this will be referred to reference committee No. 5. Hearing no objection, it is so ordered.

The Chair will recognize Dr. Burt.

Dr. Burt: I have a letter from Dr. McLaughlin to Dr. Helenbolt, regarding psychiatrists. They are having a problem regarding welfare psychiatric patients. It seems to be mandatory that these patients, after being checked by the psychiatrist, be sent to Jamestown or to Bismarck. However, quite a few people could be taken care of in private hospitals. Dr. McLaughlin feels that this is a discriminatory situation. He wishes someone to do what could be done about this. I move that this be referred to the floor as new business and disposed of in the proper committee.

Dr. Evans: I second this motion.

Speaker Dahl: All in favor? All ayes. Motion passes. Unless there is an objection, this will be referred to committee No. 5, Dr. Burt, Chairman. Hearing none, it is so ordered.

Dr. Burt's Committee also has under its jurisdiction, as stated in the Handbook, the Review of the Ad Hoc Committee on Heart Disease, Cancer and Stroke. You may not be aware of this, but Dr. Lee Christoferson and his committee have done an outstanding job. They have completed a Feasibility Study, and that is the application that will be acted upon by Dr. Burt's Committee. As you will recall, the action of this House, coming out of Dr. Miles' Committee a year ago, was to advise the Council to set up a Foundation which in turn was to make an application for a Feasibility Study for Heart Disease, Cancer and Stroke; the Feasibility Study to be accepted before an Operational Grant was applied for. This Feasibility Study is at the present time being checked and final action will be made in Washington by HEW. I wanted to give you a little background as far as the new delegates are concerned. Dr. Burt's Committee will inform us of this on Sunday.

Dr. Dunnigan: I wish to introduce a resolution to the effect that the interim meeting of the House of Delegates during the legislative year be held at a time primarily selected by the Speaker of the House, so that the meeting would better serve the purpose of handling proposed legislation. In other words, the resolution would give the Speaker of the House the prerogative of setting the time for the interim meeting.

Speaker Dahl: Are you suggesting that this be referred to some reference committee for action?

Dr. Dunnigan: Yes, I feel that such an idea should be considered by the House. I move that the time of the interim meeting be referred to a committee for study.

Dr. Lindelow: I second the motion.

Dr. Uhmer: I would remind Dr. Dunnigan that on page 150 of the Handbook the resolution from the Stutsman County Society pretty well takes care of that. It is a subject related to the interim meeting.

Dr. Dahl: Is there any other discussion? All in favor? All ayes. Motion passes. Because the resolution from the Stutsman County Society is referred to committee No. 6, I will also refer this to committee No. 6, unless there is an objection from the floor. Is there any other business to be brought up at this time?

Dr. Toomey: According to protocol, I am holding an "Open House" in this room tonight at 6:00 p.m. I wish to invite all members of the House of Delegates, Councillors, Officers and any guests you may wish to bring. You may also bring your wives.

(At this time, Dr. Buckingham advised the reference committees of meeting rooms available. The various chairmen stated the time and place of committee meetings).

Dr. Richardson: Are all members of the Association allowed to go to the reference committee hearings?

Speaker Dahl: Yes, anyone who has paid his dues may appear at any reference committee meeting. I hope in the future we will have an agenda to go out to the delegates. Is there any new business?

At this time I would like to call on our Secretary, Dr. Buckingham, to read the names of the Nominating Committee.

Dr. Buckingham: The members of the Nominating

Committee are: Dr. C. J. Glaspel, Chairman; Dr. R. E. Mahowald and Dr. Keith Foster.

Dr. Dahl: The Chair will recognize Dr. Ulmer regarding COMPACT-AMPAC.

Dr. Ulmer: It has been announced that at the luncheon tomorrow noon, Mr. Thomas Kleppe would be our speaker. However, Mr. Kleppe had to cancel his appearance because of a hearing being held on REA in Washington this day. At the moment we have no plans for this luncheon; officially it was cancelled. However, there is a possibility that we may have a program and if this can be arranged, we will announce this in time for you to attend.

Speaker Dahl: Are there any communications from the Council to be brought before the House?

Let us try to make it a point to check the exhibits. The exhibitors pay the biggest part of the expenses for the Convention. I would also suggest that everyone take cognizance of the specialty meetings being held tonight, and some are also having luncheons today.

Our next meeting of the House of Delegates is at 1:00 p.m. Sunday. This is an important session and we want to give everyone adequate time to present their case, but we still have to get back to practice by Monday morning. I implore you to be here before 1:00 on Sunday so that we may start promptly and perhaps be able to finish at 4:00 p.m. The members of the Council have to meet after the House of Delegates.

Dr. Richardson: I move that this meeting adjourn.

Dr. Hankins: I second this.

Speaker Dahl: All in favor? All ayes.

Adjournment: 9:35 a.m.

Speaker Dahl called the meeting to order at 1:00 p.m., at the Prince Hotel, Bismarck, North Dakota.

Dr. Richard J. Ulmer, Chairman of the Credentials Committee, reported there was a quorum present.

Secretary Buckingham called the roll and the following delegates responded:

Drs. H. A. Norum, Fargo; M. H. Poindexter, Fargo; A. C. Burt, Fargo; Roy Kulland, West Fargo; Paul Beithon, Wahpeton; B. J. Amidon, Fargo; R. J. Ulmer, Fargo; G. H. Hilts, Cando; R. D. McBane, Devils Lake; C. R. Miller, Bottineau; J. A. Sandmeyer, Grand Forks; G. L. Countryman, Grafton; K. S. Helenbolt, Grand Forks; H. W. Evans, Grand Forks; W. T. Powers, Grand Forks; B. J. Clayburgh, Alternate, Grand Forks; L. A. Giltner, Minot; G. R. Richardson, Minot; B. Jayapathy, Minot; A. M. Cameron, Minot; W. B. Huntley, Alternate, Minot; J. W. Goven, Valley City; O. V. Lindelow, Bismarck; J. J. Moses, Bismarck; M. A. K. Lommen, Bismarck; R. J. Dunnigan, Bismarck; Edmund Vinje, Hazen; W. C. Riecke, Bismarck; P. R. Gregware, Alternate, Bismarck; E. O. Hieb, Jamestown; J. V. Miles, Jamestown; D. E. Skjei, Williston; W. C. Hanewald, Dickinson; R. E. Hankins, Mott; Kenneth Wakefield, Alternate, Cooperstown.

There were 31 delegates and 4 alternates present. Others present were: Drs. G. W. Toomey, V. J. Fischer, C. H. Peters, C. J. Glaspel, Amos Gilsdorf, R. C. Painter, R. E. Mahowald, W. R. Fox, Dean Strinden, R. W. McLean, Frank DeCesare, J. M. Van der Linde, R. S. Larson, C. J. Klein, J. J. Moses, T. E. Pederson, and Mr. Lyle Limond and Mr. W. David Coyner.

A motion was made by Dr. Countryman, seconded by Dr. Hankins, that the reading of the minutes of the first session be dispensed with. Motion passed.

Speaker Dahl at this time welcomed Dr. Glaspel, a past president, to the meeting and introduced him to the members of the House.

Dr. Glaspel: Thank you. I find these meetings very stimulating to me. I have not missed any since 1920, except in 1926 when I was out of the state. As I sit here this afternoon, I am amazed that there are so many people whom I know by their faces, but have a little difficulty remembering their names. The other day I thought I would not attend but by Friday I had decided to come. One never knows how many meetings one is going to make in the future, and I am always happy to meet people that I only see once a year.

Speaker Dahl: Thank you, Dr. Glaspel. It is amazing to me to hear someone say that they have not missed a meeting for about 40 years.

The highlight of our state meeting is to recognize those few individuals from our profession who have dedicated fifty years of their lives to the profession and to patients they have served so long and so well.

The first recipient of the 50 Year Honor Award this year has been in private practice in Bismarck since July, 1923. Dr. Diven is still very active in this practice. He was born Dec. 13, 1889 at Atlantic, Iowa. He graduated from the University of Iowa Medical School, Iowa City, Iowa, in 1917, and was licensed in North Dakota in July, 1923.

Dr. Peters and Dr. Painter would you please escort Dr. Diven to the front of the room, and Dr. Fischer will present him with his pin and Certificate.

Dr. Fischer: Dr. Diven, it is my extreme pleasure, acting in behalf of the North Dakota State Medical Association, to present you with this Certificate of Distinction for 50 Years in the practice of medicine. This entitles you to join the 50 Year Club. And, I wish to present you with this pin for 50 years of service to your profession. I feel we are all indebted to you for the work you have done these many years. Thank you. We are very happy to have you here today.

Dr. Diven: Thank you very much. I hope it is more for the meritorious work I have done, than the endurance.

Speaker Dahl: This is a real pleasure. Dr. Dunnigan, Chairman of the Publicity Committee, asked me to take care of this and of getting a photograph of Dr. Diven. Mrs. Fremming and Mrs. Kohler, the staff at the state office, and three photographers tried for several days to get in touch with Dr. Diven and finally got hold of him yesterday but he said he was too busy to have his picture taken. As a result, he did not have his photograph taken.

Dr. G. Arthur Larson, retired, of Fargo, N.D. is not attending the meeting today. He is on a trip right now. Dr. Larson was born Oct. 3, 1889 at Minneota, Minn. He graduated from the University of Minnesota, Minneapolis, in 1917 and was licensed in North Dakota in July 1920. He was in active practice in Fargo until the time of his retirement in 1965.

It has been the suggestion of our very wise Executive Secretary, that when Dr. Fischer makes his first appearance which will be at Fargo in the fall, he will personally present this to Dr. Larson.

The representative of the Board of Medical Examiners, Dr. Fortney, was not able to attend this meeting. Dr. Cleary provided the report which you will find in front of you.

Dr. Buckingham: This is a letter addressed to me by Mrs. Fremming of the Executive Secretary's office:

"We have been advised by A. C. Fortney, M. D., Chairman of the North Dakota State Board of Medical Examiners that he is uncertain as to whether or not he will be able to be present at the second session of the

House of Delegates to make the presentation of the above referred to activities.

J. W. Cleary, M.D., Secretary of the Board, has respectfully requested that a report of the activities be prepared for your presentation to the members of the House of Delegates, so that it may become a permanent record of the proceedings, and that copies be prepared for each member.

Please advise this office if this will be satisfactory in the event Dr. Fortney is not able to appear."

Speaker Dahl: The chair will entertain a motion that this will be incorporated in the minutes.

Dr. Beithon: I move that this report be included in the minutes.

Dr. Sandmeyer: I second the motion.

Speaker Dahl: All those in favor? All ayes. Motion passed.

Report: Activities of the North Dakota State Board of Medical Examiners for the Year 1966

1. During 1966, 52 candidates were examined by the State Board of Medical Examiners.
   A. 25 applied for licensure by examination.
      1. 2 were U.S. citizens and were awarded permanent licenses.
      2. 19 were non-citizens and were issued temporary licenses, which can be replaced by permanent licenses when U.S. citizenship is obtained.
      3. 4 failed.
      4. These 25 applicants were from:
         United States (2)
         Canada and Iran (2 each)
         British Isles (7)
         Philippines (5)
         China (3)
         Rhodesia, Egypt, Turkey and Spain (1 each).
   B. 27 applied for and were licensed by reciprocity and/or endorsement.
      1. Licensure was based on:
         a. National Board Certificates (7)
         b. Minnesota License (6)
         c. Pennsylvania and Kansas Licenses (3 each)
         d. Oregon, Ohio, Rhode Island, Washington, California, Iowa, Georgia and Nebraska Licenses (1 each).
   C. Residence and practice in North Dakota of 48 given temporary or permanent licenses:
      1. 1 is still in residency
      2. 1 is with the U.S.P.H.S.
      3. 4 never took up residence in North Dakota
      4. 4 practiced a few months then left the state
      5. 38 are still in practice in North Dakota.

2. Other activities of the Board of Medical Examiners: Other important activities of the Board consist in reviewing the status of several of our practicing doctors who have problems that require the Board to check on their conduct professionally and morally at their sites of practice. These individuals are being constantly observed and reports from reputable sources come to the Board prior to the meeting at which these doctors are to appear before the Board for verbal examination. In some instances we have suspended their license especially where they have left the state and the others are quite aware of the fact that we are keeping track of them and for the most part of them, they are doing fairly well. At the end of the verbal examination by members of the Board, a determination is made as to a follow-up and what action will be taken. The individual is notified then, in writing, by the Secretary of the Board's decision. These are the situations that constitute one of our "headaches."

Respectfully submitted,
(Signed) A. C. Fortney, M.D., *Chairman*
North Dakota State Board of
Medical Examiners

Speaker Dahl: At this time we have the real highlight of the afternoon. This only happens at one of the two sessions that we have. It gives me a great deal of pleasure to present a lady who has done a tremendous lot of work for the Auxiliary this past year. She has done a terrific job for the Auxiliary and when they do that, they are actually doing a terrific job for the Medical Association.

Let us rise and welcome Mrs. Hieb, President of the Woman's Auxiliary.

President's Address, Woman's Auxiliary

It is indeed my pleasure to represent the Auxiliary here today and address this House of Delegates. Upon receipt of your invitation my good husband promptly advised me to be brief, for he assured me that by 1 p.m. on Sunday you would all be mighty weary and so, brief, I promise to be.

One of the major purposes of the Auxiliary program is to interpret the Medical Profession and its stand on social issues to a misunderstanding public. This is not done exclusively by verbal expression but it is demonstrated through service. We are a working organization and support a program that embodies a wide variety of projects for the promotion of health and good will.

Let me tell you of some of the projects your North Dakota Auxiliary has supported and started this past year.

Health Career Clubs are sponsored actively in several districts, allowing many young people the opportunity to become acquainted with professions in all medically oriented fields. Our members are working in the training of Candy Stripers for volunteer work in hospitals. This interest in health careers is substantiated by cooperation from every district in contributing to our 17 year old Medical Auxiliary Student Loan Fund. This year's donation of $2764.00 brings its total worth to $37,668.00. We are justly proud of this. The loans from this fund at our University of North Dakota Medical School are highly desirable since other available monies carry a 6 or 7% interest rate and we allow 2½% and draw no interest until after graduation.

A.M.A.-E.R.F. is high on our priority list. Fund raising, donations and memorials have allowed us to send $1,738.50 for 1966-67. The great bulk of this contribution has been designated to the University of North Dakota. With the advent of medicare and medicaid we are more acutely aware of legislation and keeping informed. A request only yesterday from National regarding immediate action on the unfavorable portions of H.R. 5710 (Social Security Amendments of 1967) will produce letters to our Congressmen from the Auxiliary.

The International Health Activities Committee is packing today the drug samples, bandages, and supplies that you patiently helped your wife bring to Convention. 25 to 30 cartons will be shipped tomorrow to World Medical Relief.

Our Mental Health Committee has been working for the establishment of four community Health Centers throughout the State. Two are in operation, and two will open this summer.

In the field of Safety, Promotion of Auxiliary sponsorship of Defensive Driver Training Courses is in progress in areas where it has not already been established.

One district sponsored a film on venereal disease education in the public school system. It was well received and was shown in physical education classes to 1500 students in grades 8 through 12.

Another district is exploring the possibility of catalyzing a Homemaker Service. This would be a "First" for our Auxiliary.

In reviewing the community service in which our membership is involved I can assure you they are all outstanding leaders in their communities. They donate their energies to fund raising for health agencies, to volunteer work in blood banks, hearing and immunization clinics and rehabilitation centers. They entertain senior citizens, aid needy families and sustain responsible positions in church and school.

1966-67 shows the Auxiliary with about seventy members less than your medical society. Team membership has not been overwhelmingly accepted by all districts since only two have instituted this practice of you doctors paying your wives' $5.00 dues to the Medical Auxiliary at the same time you pay your own dues. I hope each member of your component societies will be interested in and encourage his wife's participation in this Auxiliary. If one is unable to actively take part in our working program, we will appreciate her financial backing. We need the support of every North Dakota physician's wife to aid our growth and stature, and promote our purpose as the closest ally in your Profession.

In closing, to each of you, my sincere thanks for your kind attention.

Speaker Dahl: I hope every delegate when they get back will give this report to their home districts that the Woman's Auxiliary has raised $37,668.00 for our Student Loan Fund.

I would like to introduce another member of the Auxiliary who will present a check to our President Dr. Fischer. It gives me a great deal of pleasure to introduce Mrs. James Moses.

Mrs. Moses: I am very proud of our women. This year $2,764.00 is donated to the Student Loan Fund. This is going up every year and your Chairman will see that it will go over the $3,000.00 mark next year, I am sure.

Dr. Fischer: On behalf of the State Medical Association, I certainly want to thank you for this tremendous check for the Student Loan Fund. If all you men could see this work and how you women are working, I think this would double. Thank you, very much.

Speaker Dahl: Thank you very much Mrs. Hieb and Mrs. Moses.

We have another past state president who just came in at this time. Will you please welcome Dr. Amos Gilsdorf.

Will you please come forward, Dr. Harwood. Dr. Fischer has a little presentation to make to you.

Dr. Fischer: At this time, Dr. Harwood, it gives me great pleasure on behalf of the State Medical Association, as well as the Woman's Auxiliary, to turn over to you the AMA-ERF fund in the amount of $3,999.09; (let's call it $4,000.00) and also the check from the Woman's Auxiliary for $2,764.00 to be used as the Student Loan Fund. I am sure that you will see to it that this will go to the proper places to do the greatest good.

Dean Harwood: It would appear that I came over just this morning just to get this money, but we have had two full sessions this week on our budget. One of the things I did yesterday was knock out all travel for myself and my staff at the School of Medicine. This check will take care of travel for members of my staff. This is very attractive to our younger people who do like to go to their meetings, and if they cannot make it on a grant, this AMA-ERF check provides a way to do so. A great deal of this Auxiliary money does come out of your pockets. If it would be of interest, I could send Lyle something for his News Letter on the number of students this money has been loaned to. As soon as it comes in, it goes out again, and has been used and re-used. Thank you very much.

Speaker Dahl: Thank you, Dean Harwood.

Dr. Buckingham: This is a letter addressed to Mr. Lyle Limond from Dr. Blasingame of the American Medical Association:

Dear Mr. Limond:

"We enclose checks representing the AMA-ERF allocations for this year to medical schools in your state, together with copies of our letters to the deans.

Contributions making this allocation possible came in 1966 from physicians throughout the United States and its possessions. The amount being distributed to all medical schools is $1,051,933.11.

We shall again appreciate your cooperation in arranging suitable presentations of these checks to the deans. If you secure publicity for the presentations, we should like to receive clippings of stories and pictures."

Sincerely,

(Signed) F. J. L. Blasingame, M.D.

Dr. Evans moved, seconded by Dr. Miller, that an item be placed in the News Letter regarding the loan fund and presentation to Dr. Harwood. Motion passed. Speaker Dahl directed the Association office to do this.

Reference Committee to Consider the Reports of the President, Secretary, Executive Secretary and Treasurer

Dr. Hankins, chairman of this committee, presented the following reports and their discussions, which were adopted section by section, and as a whole.

1. Report of the President. This report was reviewed and approved. The committee noted with approval the election of Dr. Clifford Peters as President-Elect of the North Central Medical Conference and registered support from the North Dakota State Medical Association in his activities. The committee did not feel that sending a single additional representative to the AMA House of Delegates meeting was feasible. They did suggest that the Council recommend to each District society that they, in turn, and if possible, send one man who would report at the subsequent meeting of the district society.

The committee commended President Toomey and thanked him for his work on behalf of the State Association. In further discussion concerning the feasibility of sending a representative from a district society to attend the AMA House of Delegates as an observer, not at the Association's expense, it was agreed that this should be arranged by the individual district societies. This portion of the report was adopted.

2. Report of the Secretary. This report was reviewed and approved. The committee noted that in the Secretary's report paragraph 1, page 17 of the Handbook was incomplete in view of the action taken at the 1966 House of Delegates, which amended the By-Laws to read that "representation in the House of Delegates is based on one delegate for each 15 paid-up members (or major fraction thereof), as of December 31st of the

previous year, together with honorary memberships." Delegates from the local societies should be seated on this basis. It was felt that the tabulation of paid memberships is superfluous except as a point of information for the district society secretaries regarding collection of dues. Mr. Limond informed the members that this year the House was seated according to the cut-off date of December 31st. This portion of the report was adopted.

3. Report of the Executive Secretary. This report was reviewed and approved. The committee called to the attention of all members, that any bills which are to be introduced to the State Legislature are to be prepared two months prior to the convening of that body, and at that time are to be submitted to the office of the Executive Secretary of the Association for duplicating and proper disposition.

Mr. Lyle Limond was commended for his continued efforts on behalf of the Association, especially in view of his recent disability. The committee wished him a complete recovery. The statement of expenses of the Association was reviewed and approved by the committee. Mr. Limond explained that it was just one of his thoughts for the future that any legislation the district societies wished to introduce should be prepared well ahead of time. The members of the Association could then be informed for their comments and suggestions before presentation to the State Legislature. This portion of the report was adopted.

4. Report of the Treasurer. This report was reviewed and approved. The committee failed to note a declaration of interest on C.D. No. 3893. Their assumption was that this Certificate of Deposit is on a 12 month basis, but would appreciate a clarification thereof by the Treasurer. Dr. Nierling stated that he would clarify on this later. This portion of the report was adopted.

The motion was made by Dr. Hankins, seconded by Dr. Powers, that this report as a whole be adopted.

Dr. Robert E. Hankins, *Chairman*
Dr. Roy E. Kulland, *Vice-Chairman*
Dr. C. Robert Miller
Dr. William T. Powers
Dr. Neill Goltz, *Alternate*
Dr. William Riecke

Reference Committee to Consider the Reports of the Council, Councillors, Special Committees and the Report of the Representative to the Blue Shield Board of Directors

Dr. Countryman, chairman of this committee, presented the following reports and their discussions, which were adopted section by section, and as a whole.

1. Report of the Chairman of the Council. The report of the Chairman of the Council was reviewed by the Reference Committee who asked if Mr. Daner had taken any action regarding the reduction of Workmen's Compensation fees for physician-employees, since they are in non-hazardous occupations. The following special observations were noted by the committee:

1—Professional and financial grievances should be left to local district societies and only referred to Council if they cannot be satisfactorily settled on the local level.

2—The Committee for Heart Disease, Cancer and Stroke is to be commended for their work and we await with interest the result of the Application for a Feasibility Study Grant.

3—The quotation of the Crippled Childrens Service Committee is noted. It seems ambiguous and we go on record as recommending that the usual customary and reasonable fees apply here as well as in all third party payments.

4—The resolution in regard to fees charged by the University of North Dakota Medical School Pathology Laboratory is noted. In general, we feel that the fees charged should be essentially the same as charged by private laboratories for similar services. If teaching material is needed, the other pathologists in the State should be asked to assist in providing the same.

This will be directed to the Council by Dr. Dahl. This portion of the report was adopted.

2. Reports of the Councillors. The Reference Committee reviewed the reports of the Councillors of all ten districts.

The report of the Councillor of the First District was reviewed and the following noted by the committee:

A. The First District turned over $7100 to establish a trust fund for loans and scholarships for students at the North Dakota Medical School.

B. The First District recommended the establishment of a basic science law. It is suggested that, if desired, the Committee on Medical Education study the matter and report at the next meeting of the House. This portion of the report was adopted. Speaker Dahl advised that the first item is a notation, the second a suggestion that, if desired by the House of Delegates, the Committee on Education study the matter and report on a basic science law at the next meeting. He added that if passed, it would be the purpose of the First District Councillor to refer this matter to the Committee on Education. Mr. Limond advised that if this goes to the Committee on Education, they should then work closely with the State Board of Medical Examiners.

The report of the Councillor of the Second District was reviewed and the committee noted that it was suggested by one of the Second District members that some form of a Utilization Review Committee be established on the District Society level. The Reference Committee was in accord with this idea. This portion of the report was adopted.

The report of the Councillor of the Third District was read and approved. It was noted by the committee that excellent scientific speakers were invited for each monthly meeting. This portion of the report was adopted.

The report of the Councillor of the Fourth District was read and approved. The committee believes that it is regrettable that the committee to establish the Ward Mental Health and Retardation Service Unit did not include a physician from the Fourth District Society. The committee applauded the interest of the Fourth District in Nursing Education. This portion of the report was adopted.

The report of the Councillor of the Fifth District was reviewed and approved. This portion of the report was adopted.

The report of the Councillor of the Sixth District was reviewed and the committee noted the success of the Heartview Foundation, a non-profit, non-sectarian organization for the treatment of alcoholism. It was the committee's belief that the endeavor of the Foundation should have the Society's appreciation. This portion of the report was adopted.

The report of the Councillor of the Seventh District was reviewed and approved. The committee commended the Seventh District for giving $6,744.10 to the Jamestown College for the use for scholarships for nursing students, and also to make loans to these students. This

portion of the report was adopted.

The report of the Councillor of the Eighth District was reviewed and accepted. This portion of the report was adopted.

The report of the Councillor of the Ninth District was reviewed and approved. This portion of the report was adopted.

The report of the Councillor of the Tenth District was reviewed and approved. This portion of the report was adopted.

3. Report of the Representative to the Blue Shield Board of Directors. This report was reviewed and approved by the Reference Committee. The following was noted by the committee:

1—Because of the frequency of introduced legislative bills which may have affected the Blue Shield program, and which were defeated, it is felt that this is a matter for greater concern and watchfulness. A full-time lobbyist should be employed.

Your reference committee is in accord with such a suggestion.

2—This committee notes that an Ad Hoc Committee was appointed to consider the desirability of the addition of lay persons to the Board of Directors of Blue Shield.

3—Due to the confusion existing as to the usual and customary fees, pertaining to Blue Shield and Medicare, the following guide-lines approved by the Blue Shield Board of Directors, were noted. Your reference committee feels this is important enough to quote verbatim:

"1—Since Blue Shield has never paid for office calls in any of its basic subscriber contracts either before or after surgery, the 'reasonable' fee determination for surgery will be on the basis of 'prevailing' fees to include the surgery and up to two weeks necessary after-care in the hospital. No allowance will be made for anticipated follow-up office calls. It would be the doctor's privilege to bill the patient for these calls and they would be paid for by the patient to him.

Should the patient require hospitalization beyond the normal two weeks post-operative period, medical calls for this additional care should be itemized on the claim and consideration for additional payment will be made by Blue Shield.

"2—Government Medicare Part B *does* allow payment for necessary office calls but does not allow 'prepayment' for office calls anticipated in the future.

Payment for surgery would be on the basis as outlined above. In addition, payment of the 'reasonable' fee for necessary office visits following surgery will be made but such visits must be itemized and billed to either Medicare Part B, or to the patient."

This portion of the report was adopted.

Dr. Countryman moved that the report be adopted as a whole. The motion was seconded by Dr. Norum. Motion carried.

Dr. G. L. Countryman, *Chairman*
Dr. R. D. McBane, *Vice-Chairman*
Dr. Henry A. Norum
Dr. B. F. Amidon
Dr. O. V. Lindelow

Reference Committee to Consider the Reports of the Delegate to the AMA, Medical Center Advisory Council, Committee on Medical Education, and the North Dakota State Advisory Council for Hospital and Related Construction

Dr. Miles, chairman of this committee, presented the following reports and their discussions, which were adopted section by section and as a whole, with the exception of the Committee on Nursing Education.

1. Report of the Delegate to the American Medical Association was reviewed and approved. It was the consensus of the committee that those items in the report that are not being considered specifically by the Reference Committee or any other committees deserve mention. The committee recommended that medical society review committees and utilization committees should be studied as to their special and proper function. It was further recommended that physician membership on hospital governing boards should be encouraged and advised. The committee suggested that the AMA Delegate be instructed to favor the continuance of the AMA Disability Insurance program in its present form.

After considerable discussion, this portion of the report was adopted.

2. Report of Representative to Medical Center Advisory Council. This report was reviewed and approved by the committee which reaffirmed its position that the State Health Laboratory be maintained in close proximity to the Medical School. This portion of the report was adopted.

3. Report of the Committee on Medical Education. This report was reviewed and approved by the committee. The committee, however, did recommend that the Millis Report be rejected in spite of the fact that the committee recognized the need for better clinical training for physicians because of the increasing complexities of medical knowledge and practice, and urged that steps be taken in the direction of improved clinical training. The committee recommended that the Delegate to the AMA be advised of this stand.

Dr. Ulmer, at the request of Speaker Dahl, enlarged on the Millis Report.

Dr. Ulmer: You have in your handbook on page 106, the report entitled "Millis Report." I mailed this summary to all committees and asked them to return this with their own feeling about it. Our reference committee here went through this item by item and thoroughly. If the Millis report should be adopted on a nation-wide basis, as stated here, the curriculum would be changed. Beyond that point, they are recommending that Medical Centers be advised to set up a continuing graduate program for doctors that would be a 3 year residency, and this would be rated in the field of medicine, psychiatry, pediatrics, medical gynecology and preventive medicine. The physician that would complete this thing would be the primary physician of the future. He would serve as the primary medical resource and counselor to an individual or family. The level of training would be on a par with that of other specialities. A 2 year graduate program would be insufficient. No medical center would be approved for medical training unless the teaching staff, related services and other facilities are judged adequate in size and quality. You can, therefore, appreciate the problems that would arise in our state. All states would have to change their licensure after finishing the 3 year program. It is quite a far-reaching step, and I am not sure we are ready to accept all this. That is the reason we recommend that this be not approved by this body. This portion of the report was adopted.

Dr. Miles submitted the following resolutions:

RESOLUTION CONCERNING DRIVER'S LICENSES FOR EPILEPTICS

The First District Medical Society submitted a resolution regarding the safety and insurability of drivers af-

flicted with epilepsy and regarding the encouragement that these same drivers seek and follow medical advice about their medical problem. This committee recommends that this resolution be referred to the Medical Advisory Committee to the State Highway Department for study and recommendations for adequate and proper legislation.

This resolution was adopted.

Dr. Miles:

RESOLUTION CONCERNING THERAPEUTIC ABORTION

The Seventh District Medical Society submitted a resolution regarding Therapeutic Abortion and this committee recommends that this resolution be adopted with the following changes:

Section 12-25-01 (b) Three physicians, one of whom may be the person performing the abortion have certified in writing their belief in the justifying circumstances and have filed such certificates prior to the abortion in a licensed hospital where the therapeutic abortion is to be performed, or in such other place as may be designated by law, and in addition, when mental health is concerned, a written report of a qualified psychiatrist will also be filed with the reports of the above three physicians.

We recommend that this resolution be sent to the Legislative Committee for introduction at the next legislative session.

A discussion followed:

Dr. Miles: The Therapeutic Abortion Bill was introduced previously and was rushed to the Committee on Maternal and Child Welfare last year. They wrestled with the problem for some time and they recommended that this be passed only with the addition of the incest and rape. This is in the report of the Committee on Maternal and Child Welfare in the Handbook. The Seventh District Society is resubmitting the resolution, carrying this another step further than the incest and rape aspect of this.

Dr. Amidon: The resolution was introduced two years ago. At that time the House instructed the Committee on Maternal and Child Welfare to come up with an acceptable proposal. A year ago we did not have one, and it was the feeling that the House was a little unhappy with us; so we came up with this proposal that the law be changed to add the incest or rape to this. This is a problem that becomes emotional. Religious feelings enter into it. It can stir up a great deal of controversy and it did in the committee meetings. We felt that if a girl hollers "rape", the judge will hold a hearing, and if the judge feels that this is rape, or she became pregnant from incest, he may decide that the abortion may be carried out. One of the problems is that you must have the medical advice of a psychiatrist. My personal feeling is such that I will vote "no" against it. There was difficulty getting this proposed bill into the legislature last year.

Dr. Dunnigan: In the proposed resolution as Dr. Miles has read it, the word "rape" is not defined here. I do not know what we are talking about. I presume the change was made from two to three physicians to make this more binding. However, this was somewhat of a slipshod type of arrangement to begin with. I am wondering if adding another doctor helped this situation. I would speak against this resolution.

Dr. Miles: The Committee wants the House to decide this.

Dr. Wakefield: In the Tenth District we thought that two people were no safeguard. I can not see how three people could be a safeguard. If all abortions could be referred to a Reviewing Board, it might not stop all abortions, but it might stop the greater majority of them.

Speaker Dahl: We will ask for a standing vote on this. All those in favor of the resolution? Ayes 19. Opposed, 14. The Ayes have it. The resolution will be sent to the Legislative Committee for further action. I would suggest that those interested in this bill make your views known to the Committee on Legislation.

4. Report of the Committee on Cancer. This report was reviewed and approved. The committee recommended that a Central Cancer Registry be established to pool information for statistical purposes, but that the follow-up be continued by the Local Registry Units. This portion of the report was adopted.

5. Report of the Committee on Nursing Education. This report was reviewed. It was the recommendation of the committee that this committee should be abolished in view of the lack of interest and because of the absence of current problems related to nursing education. This portion of the report was not adopted.

6. Report of the Committee on Maternal and Child Welfare. In view of the action taken by the committee on the Resolution on Therapeutic Abortion the committee agreed that no further comment need be made. This portion of the report was adopted.

7. Report of the Committee on Foreign Trained Physicians. This report was reviewed and approved. The factual study was noted with interest by the committee. This portion of the report was adopted.

8. Report of the Committee on Diabetes. This report was reviewed and approved. This portion of the report was adopted.

9. Report of the Committee on Crippled Children. This report was reviewed and approved. This portion of the report was adopted.

10. Report of the Committee on Mental Health. The committee noted that no meetings had been held by this Committee.

11. Report of the Committee on Aging and Rehabilitation. This report was reviewed and approved. This portion of the report was adopted.

12. Report of the Liaison Committee to the North Dakota State Bar Association. It was noted by the committee that no meetings were held and that no contact had been made with the North Dakota State Bar Association.

13. Report of the Committee on Liability Insurance. It was noted by the committee that no meetings had been held.

14. Report of the Committee on Constitution and By-Laws. This report was reviewed by the Reference Committee and it was noted that no meetings were held in 1967 at the time the report was submitted. It was also noted that two resolutions have been submitted by this Committee covering amendments to the Constitution and By-Laws, and that these amendments have been referred to the Resolution Committee for action. This portion of the report was adopted.

15. Report of the Liaison Committee to the North Dakota Dental Association. It was noted that there had been no special activities of this committee.

16. Report of the Committee on Cardiovascular Diseases. It was noted that no meetings were held by this committee during 1967.

17. Report of the Medical Advisory Committee to the State Highway Department. This report was reviewed and approved. This portion of the report was adopted.

18. Report of the Representative to the Crippled Chil-

drens Service Division of the Public Welfare Board was reviewed and approved. This portion of the report was adopted.

19. Report of the Committee on AMA-ERF. This report was reviewed and approved. It was noted that a plea was made for increased support of the Medical School at Grand Forks from the doctors in the state. This portion of the report was adopted.

20. Report of the Committee on Medicine and Religion. This report was reviewed and approved. This portion of the report was adopted.

21. Report of the Committee on Emergency Medical Service. This report was reviewed and approved. This portion of the report was adopted.

Dr. Miles moved that the report be adopted as a whole, with the exception of the motion regarding the Committee on Nursing Education. Motion seconded by Dr. Ulmer and carried.

Dr. J. V. Miles, Jr., *Chairman*
Dr. R. J. Ulmer, *Vice-Chairman*
Dr. H. W. Evans
Dr. A. Malcolm Cameron
Dr. M. J. E. Johnson

Reference Committee to Consider the Reports of the Standing Committees, Except Committee on Medical Education and Committee on Medical Economics

Dr. Lommen, Vice-Chairman of this committee, presented the following reports and their discussions, which were adopted section by section and as a whole.

1. Report of the Committee on Necrology and Medical History. The Reference Committee reviewed and approved this report and commended Dr. Boerth and his committee for the great amount of work involved to organize and assemble the details and accomplishments of our deceased fellow physicians. The members of the House of Delegates were asked to stand for a moment of silence in respect for the passing of Drs. A. E. Hetzler, F. B. Schleinitz, T. F. Gibson, A. F. Jensen, F. E. Anderson, C. M. Graham, A. C. MacDonald, H. A. Bowes and E. A. Weible. A moment of silence adopted this report.

2. Report of the Committee on Public Health. This report was reviewed and notice was taken that there had been no meeting of this committee during 1967.

3. Report of the Committee on Public Relations. This report was reviewed and approved. This report was reviewed also with interest, particularly their recommendation for the merger of the Committees on Legislation and Public Relations because of their closely related functions. The Reference Committee agreed with this principle. It was noted that a resolution is being considered to come before this session that would reorganize the committees of this Association into five commissions. The committee added that if this resolution is accepted, the principle will be followed. If it was not, the Reference Committee would bring up a proposed change in the By-Laws later in the session. This portion of the report was adopted.

4. Report of the Committee on Official Publication. This report was reviewed and approved. It was noted that no meeting of the Committee had been held during the past year and that the Chairman, Dr. Cleary and his Committee had recommended that this Committee be dissolved because of lack of function. The Reference Committee agreed that this should be done. The committee stated further that if the resolution on the establishment of commissions is adopted, and the By-Laws changed subsequently, the recommendation will be accomplished. This portion of the report was adopted.

5. Report of the Committee on Legislation. This report was reviewed and approved. The Reference Committee expressed their concern with the increasing degree of socialization in medicine as outlined in the report of the Committee, and added that the Association is grateful to Dr. Buckingham and his committee for their efforts and the time spent in doing their jobs during the recent legislative session. The Reference Committee noted that the matter of the Abortion Law was being reconsidered by the Resolutions Committee. This portion of the report was adopted.

Dr. Lommen moved that the report be adopted as a whole. The motion was seconded by Dr. Huntley and carried.

Dr. M. A. K. Lommen, *Chairman*
Dr. M. H. Poindexter, Jr., *Vice-Chairman*
Dr. B. Clayburgh, *Alternate*
Dr. John Goven
Dr. W. B. Huntley, *Alternate*
Dr. K. Wakefield, *Alternate*

Dr. Dahl advised the House at that time that two Reference Committees still to be heard had considerable material to bring before the House. He called a 10 minute recess to reconvene at 2:45 p.m.

Dr. Dahl called the Second Session of the House of Delegates to order at 2:45 p.m.

Reference Committee to Consider the Reports of the Committee on Medical Economics, the North Dakota Advisory Council for Hospital and Related Construction and the Ad Hoc Committee on Heart Disease, Cancer and Stroke

Dr. Burt, chairman of this committee, presented the following reports and their discussions, which were adopted section by section, as amended, and as a whole.

1. Report of the North Dakota Advisory Council for Hospital and Related Construction. The Reference Committee reviewed and approved this report. It was noted by the Reference Committee that two meetings of the Council had been held during the year. At the June, 1966 meeting they reaffirmed their policy to produce 46% of the construction costs for hospital and convalescent home care if and when funds were available. At the December 1966 meeting it was the decision of the Council that if money became available in the next fiscal year, it would be used to support the Bethel Lutheran Home for the Aged in Williston, North Dakota. This portion of the report was adopted.

2. Report of the Ad Hoc Committee on Heart Disease, Cancer and Stroke. This report was reviewed and approved. It was noted by the Reference Committee that this particular committee had been appointed by the President, Dr. Glenn Toomey in November, 1966 and the first meeting held on December 9, 1966. It was also noted that T. H. Harwood, M.D., Dean, School of Medicine, University of North Dakota was selected as program coordinator and the fiscal agent was declared to be the Medical Center, University of North Dakota, Grand Forks, N. D. In further review, the Reference Committee noted that, in accordance with the directive of the House of Delegates, this Committee developed an application for the Regional Medical Program Planning Grant of Heart Disease, Cancer and Stroke for the North Dakota region. It was felt by the Reference Committee that the work had been well done and the volume

of research and work involved in this planning segment should be commended. It was also felt by the Reference Committee that this portion of the report should be accepted as filed. This portion of the report was adopted.

Dr. Burt advised the House concerning another item of business to be resolved by his committee—a letter from Dr. B. E. McLaughlin of Grand Forks to Dr. Helenbolt referable to in-hospital psychiatric care following psychiatric evaluation. He felt that discrimination existed inasmuch as patients, after diagnosis, were required to go to the State Hospital at Jamestown, because of a law which governed the public welfare segment of our economy. This was investigated and found to be in fact only a regulation which has been utilized over a period of years to the point where people believe it is law. It is felt that this is a Medical Economics Committee matter and the Reference Committee felt that it should be handled by them. This portion of the report was adopted.

3. Report of the Committee on Medical Economics. The report was reviewed by the Reference Committee who noted that the committee had been extremely active during the past year and had performed a great service to the medical profession in the state. The Reference Committee commended the members of that committee for their work with the exception of the solution of the Crippled Childrens Department of the Welfare Board. It was noted that this will be handled later in a resolution.

A discussion followed:

Dr. Burt: It became apparent that there was considerable interest in Title 19 and also the Crippled Childrens Service and because of this, hearings were held at 12:30 in the Princess Room of the Prince Hotel, May 6, 1966. All specialists involved in this matter were represented; namely, the pediatricians, the anesthesiologists, the ophthalmologists, plastic surgery, and orthopedists. Much discussion ensued concerning the Crippled Childrens Service and the implication of Title No. 19.

Dr. John Rawland testified before the committee to the effect that:

1—His services as far as the crippled children were concerned were not voluntary, as the Crippled Childrens Service would have one to believe.

2—He indicated that he was only being paid 60% of his usual and customary fee. He would prefer not to do this work but as a matter of public service, he has not refused to do so, and he assured me that he spoke for all the anesthesiologists in the state.

The ophthalmologists were not represented at the meeting but in private conversation a number of them indicated strongly that they were in favor of the usual and customary fees for their work with the Crippled Childrens Service.

Doctor Lamb expressed his opinion as far as plastic surgery was concerned, and he indicated that he could see no reason why Crippled Childrens Service and Title No. 19 patients should not be the same as any other medical group.

The pediatricians were especially adamant in their comments concerning this program, as were the general practitioners, who, apparently over a period of years have obtained no payments for treatment before and after specialist care. It was particularly brought out at this time that there should be a free choice of physician as far as Crippled Children Service was concerned, and it also became apparent in the discussion that many peculiar practices existed, such as having to return to a specialist, 75 or 100 miles, simply for a urinalysis or hemoglobin determination.

There seemed to be no question about an unaniminity of opinion regarding these matters that patients involved in Title No. 19, of which the Crippled Childrens program is a part, should be treated in no manner different from any other group.

Dr. Pray discussed at some length, the origin of the Crippled Childrens Service and the facts are that this is a product of the depression of the 30s, and undoubtedly should have been discontinued long since.

It was the feeling of this Committee, after the hearings, that the Medical Economics Committee should, as soon as possible, negotiate with the Welfare Department concerning the problems of both the Crippled Childrens and Title No. 19, which according to law, are one and the same.

A resolution was presented to this reference committee, initiated by Dr. Wayne Le Bien of Fargo.

RESOLUTION

RESOLVED, that the House of Delegates of the North Dakota State Medical Association request that the Medical Economics Committee of the State Association consider and make appropriate plans for reviewing the fee schedules of the North Dakota Crippled Childrens Service (Department of Public Welfare), and insist that this agency accept the fee schedules as suggested in the October, 1966 Relative Value Guide of the North Dakota State Medical Association, plus a conversion factor that will allow a payment of the usual and customary fees of the physician-recipient; and,

BE IT FURTHER RESOLVED, that with the implementation of additional public assistance programs such as Title No. 19, it is paramount that similar recommendations for fees he consistent throughout all of the state welfare agencies; and

BE IT FURTHER RESOLVED that the Medical Advisory Committee to the Public Welfare Board regarding Title No. 19, be encouraged to act forcefully in their negotiations with the Welfare Board.

I move the adoption of this Resolution.

Dr. Richardson: I second this motion.

Dr. Dahl: Is there any discussion?

Dr. Vinje: The problem we have run into in this matter is that when we go to the Welfare Board and ask them why it is necessary for this youngster to be sent down to a pediatrician in Bismarck for routine tests, and why they can not have some of this work done at home to save some money, the answer is that because of federal funds being involved, they have nothing to say about this. If we are going to have Title No. 19 added on to this, and designate the physician, I think this is a very serious matter. I think before we get involved in this Title No. 19, that the choice of physician be emphasized.

Dr. Lindelow: The C.C.S. has accepted the Guide, but on their own conversion factor.

Dr. Miles: In the report of the Committee on Crippled Children in the Handbook on page 127, third paragraph, it states "it was pointed out to the members of the committee that CCS is now working on state funds."

Dr. Burt: I looked up the law yesterday and the CCS is part of Title No. 19.

Speaker Dahl: Is there any further discussion? All in favor of the resolution presented by Doctor Burt and his Committee? All ayes. Motion passed.

Dr. Burt: Another item of business came up before the Committee, involving testimony by Dr. Huntley of Minot, who presently is Chairman of the Rates and Planning Committee of the North Dakota Blue Shield. It becomes more apparent that whether desirable or not, the Medical Economics Committee of the State Association and the North Dakota Blue Shield are confronted with the same problems, and a liaison between the two organizations is most essential. As a result, he suggested that the Chairman of the Medical Economics

Committee of the State Association should be an ex-officio member of the Rates and Planning Committee of Blue Shield, and that the Chairman of the Rates and Planning Committee should automatically, be on the Medical Economics Committee of the State Association. Your reference committee strongly suggests that this be implemented.

I move the adoption of this portion of the report.

Dr. Skjei: I second the motion.

Speaker Dahl: Is there any discussion? All those in favor? All ayes. Motion passed.

Dr. Burt: There is one last item of business that I wish to present as a matter of information only regarding Title No. 19. This does not appear in the law. However, it has appeared as a regulation from HEW which says that a vendor of medical service who is unhappy with the carrier designated by the Social Security Department can complain and substantiate his complaints to HEW, and they will in turn attempt to improve the situation.

This may become important to us in the future, and it is desirable for everyone to know that this mechanism is available, which would indicate that if the State Welfare Department does a poor job with administering Title No. 19, the component district societies, as well as the State Association, can make a complaint to the proper authorities which may produce results.

I move the adoption of the report as a whole.

Dr. Moses: I second this.

Dr. McBane: I just wonder if I might go back and talk about Title No. 19 and the CCS. I would like to speak in support of Dr. Vinje's ideas. This might very well expand into a situation that only Board Certified physicians would be allowed to look after patients covered under Title No. 19. I just wonder if another amendment might not be put in this resolution just passed, to re-emphasize what has been stated.

Speaker Dahl: I would ask Dr. Hilts to give us some light on this subject.

Dr. Hilts: This will be taken care of, Dr. McBane, we hope. The committee that is meeting on this will emphasize the free choice of physician. I can talk to you personally on this later, if you so wish.

Dr. McBane: I would really like to know the mechanism by which this is going to be taken care of. Some of the men here, I am sure, would also like to know how this is going to be done.

Dr. Burt: We have all negotiated with the Welfare Board over the years. As a result, we have a committee now that had to be appointed by law by the President of the State Medical Association. The committee is called the Medical Advisory Committee to the Public Welfare Board. This, in effect, is a Medical Association Committee, instead of a "yes" committee which has been available to CCS. This committee will be forceful. I think the Medical Economics Committee is going to have to do their bit on this too.

Speaker Dahl: The Chair will recognize Dr. Buckingham.

Dr. Buckingham: I want to speak to the rest of the group who were not at the meeting. This free choice of physicians was brought up by the specialists and passed by the specialists.

Dr. Miles: As Lyle Limond has told us; first of all, we are physicians, and I think we will have to forget our little differences between specialties. We have to forget what each one of us is interested in and work together on this, if we are going to combat the Public Welfare segment now. I have to put aside what I think on pediatrics, and help others accomplish what they want also. I can not believe that we can not all work together and all come out on top of this. If the CCS states that the general practitioner is no help in this program, we are not exactly working together on this.

Dr. Skjei: I am the representative from the North Dakota State Medical Association to the Medical Advisory Committee to Crippled Childrens Service. I am appointed by the North Dakota State Medical Association. I do not know if it is necessary for the physician of the Medical Advisory Committee to handle all of these things. This idea of the general practitioner not participating in this program has been brought up for many years. Their argument is this that the scope of CCS does not lend itself to what the general practitioner can do. Most of these are procedures which the general praetitioner is not capable of doing. There is a possibility that he can participate if the specialist will refer the child back to him to do what he can take care of. This has been sent to all the physicians in the state and I think they will start doing these things. A lot of things are being done for the crippled child that the general practitioner could do if the specialist will remember to refer the child back to him.

Dr. Wakefield: I thought that all Dr. McBane wanted to know was how the free choice of physician was going to be implemented, in this thing.

Speaker Dahl: Do you have a motion, Dr. McBane? If you do, the first motion would have to get this back on the floor which would take a two-thirds majority vote.

Dr. McBane: I agree with all of the things that Dr. Burt said in his report. However, I feel that the House should be a little stronger than this resolution seemed to be. I would make a motion to reconsider the action we took on Dr. Burt's report on the resolution from the First District.

Dr. Wakefield: I second the motion.

Speaker Dahl: All in favor of the reconsideration? All ayes. The resolution is on the floor for reconsideration.

Dr. Burt: (Again read the Resolution).

Dr. McBane: In the resolution, in the second paragraph, at the end of the last sentence ending "all of the state Welfare agencies", I would move an amendment to read "recognizing a free choice of physician".

Dr. Vinje: I second this.

Dr. Miles: I am not sure what is meant by "free choice of physician".

Dr. McBane: The idea is that if this were so, and it was decided that the child must go to a specialist or pediatrician, the physician could send the patient but the child would still be his patient. It would not be necessary to go 100 miles for a urinalysis. He could go back to his regular doctor. I believe we should all work together on this thing.

Speaker Dahl: All in favor of inserting the amendment in the resolution to read "free choice of physician"? All ayes. Resolution is amended.

All those in favor of the resolution as amended with the new language? All ayes. Resolution is passed as amended.

Dr. Burt moved that the report, as amended, be adopted as a whole. The motion was seconded by Dr. Moses and carried.

Dr. A. C. Burt, *Chairman*
Dr. J. J. Moses, *Vice-Chairman*
Dr. G. H. Hilts
Dr. W. W. Nelson, *Alternate*
Dr. D. E. Skjei
Dr. G. R. Richardson

Report of the Reference Committee on Resolutions To Include New Business

Dr. Vinje, chairman of the committee, submitted the following resolutions:

RESOLUTION No. 1

WHEREAS, the members of the North Dakota State Medical Association, attending the Eightieth Annual Meeting of this Association in Bismarck, have enjoyed the hospitality and consideration of this great city, and

WHEREAS, the Mayor of Bismarck and his associates; the press, radio, hotels and businessmen have contributed to make this session a memorable one,

NOW, THEREFORE, BE IT RESOLVED that the House of Delegates express its appreciation by directing a copy of this Resolution to the Honorable Mayor of Bismarck, and the press.

This resolution was adopted.

RESOLUTION No. 2

WHEREAS, the members of the North Dakota State Medical Association anticipate the increase in scientific knowledge to be gained from attentance at various exhibits, and

WHEREAS, the Exhibitors have shown unusual effort and ingenuity in developing their exhibits and thereby, adding to the scientific interest at this meeting,

NOW, THEREFORE, BE IT RESOLVED that the North Dakota State Medical Association extend to these Exhibitors our hearty welcome and thanks, and

BE IT FURTHER RESOLVED that a copy of this Resolution be forwarded to each Exhibitor.

This resolution was adopted.

RESOLUTION No. 3

WHEREAS, the ladies of the Woman's Auxiliary to the North Dakota State Medical Association, through devoted enthusiasm and continuous efforts over the years have contributed diligently to the Medical Auxiliary Student Loan Fund of the Medical School of the University of North Dakota, and more recently to the American Medical Association Education and Research Fund, and

WHEREAS, these funds have enabled many medical students to complete their medical education, and

WHEREAS, the local Auxiliary Societies throughout the State have participated in many other commendable projects which reflect credit to their organization and to the Medical Profession,

NOW, THEREFORE, BE IT RESOLVED that the House of Delegates of the North Dakota State Medical Association convey to the Woman's Auxiliary our appreciation and thanks for their excellent work, and

BE IT FURTHER RESOLVED that a copy of this Resolution be directed to Mrs. Edwin O. Hieb and her successor.

This resolution was adopted.

RESOLUTION No. 4

WHEREAS, the present Committee System has become quite cumbersome and redundant, and

WHEREAS, this is a small society with few members available to act on Committees, and

WHEREAS, many committees over the years have been appointed which now no longer serve any useful purpose,

NOW, THEREFORE, BE IT RESOLVED that Chapter VIII of the By-Laws be amended to eliminate all present committees and be replaced with a Commission System set-up as follows:

FIVE COMMISSIONS ARE PROPOSED, NAMELY:

A. Commission on Medical Services.
B. Commission on Scientific Medicine.
C. Commission on Communications.
D. Commission on Allied Organizations.
E. Commission on Legislation and Governmental Relations.

Taking into consideration the Association's present Standing and Special Committees, plus our representatives on Governmental Councils or Committees, the Commission structure would be as follows:

COMMISSION ON MEDICAL SERVICE: covering:
A. Emergency Medical Service.
B. Foreign Trained Physicians.
C. Crippled Children.
D. Aging and Rehabilitation.
E. Liability Insurance.
F. Mental Health.
G. Public, Rural and School Health.

COMMISSION ON SCIENTIFIC MEDICINE: covering:
A. Scientific Program.
B. Arrangements for Annual Session.
C. Medical Education.
D. Nursing Education.
E. Cancer.
F. Diabetes.
G. Cardiovascular Diseases.
H. Multiple Sclerosis.
I. Maternal and Child Welfare.

COMMISSION ON COMMUNICATIONS: covering:
A. Public Relations.
B. Official Publication.
C. Necrology and Medical History.
D. AMA-ERF.
E. Physician's Placement Service.
F. Constitution and By-Laws.

COMMISSION ON ALLIED ORGANIZATIONS: covering:
A. North Dakota Hospital Association.
B. North Dakota Pharmaceutical Association.
C. State Bar Association of North Dakota.
D. North Dakota State Dental Association.
E. Woman's Auxiliary.
F. Medicine and Religion.
G. North Dakota State Nurses Association.

COMMISSION ON LEGISLATION AND GOVERNMENTAL RELATIONS: covering:
A. Legislation, Medical Economics and Governmental Agencies.
B. Medical Center Advisory Council.
C. Governor's Health Planning Committee.
D. State Health Council.

The President of the Association would appoint the Committee Chairman as well as the members. Two Councillors would be assigned to each Commission in order to assure better communications.

I move the adoption of this Resolution.

Dr. Beithon: I second the motion.

Speaker Dahl: The Chair would state at this time that although it is my opinion that our first session was Friday, and as this does not amend the Constitution but amends the By-Laws, you could be making a permanent amendment to the By-Laws this afternoon. Since there is a controversy about amending the By-Laws, the Chair would rule at this point that we consider this the first reading of this amendment and we will have the final action at next year's session; unless there is a request from the floor to the contrary.

All those in favor? All ayes. Motion passed. This resolution will then be up for final action in the next Annual Session in 1968.

RESOLUTION No. 5

WHEREAS, the House of Delegates of the North Dakota State Medical Association has held an Interim Meeting in early December prior to the opening of the North Dakota Legislature, and

WHEREAS, the pertinent legislation the North Dakota Medical Association is interested in, is usually not in bill form, or introduced in the Legislature before the third week in January of the legislative year, and

WHEREAS, the Interim Meeting of the House of Delegates could be more effective if held in Bismarck at such a time to provide more accurate awareness of all bills submitted;

NOW, THEREFORE, BE IT RESOLVED that the Interim Meeting of the House of Delegates of the North Dakota State Medical Association be held on such a date as deemed wise by the President of the State Medical Association, and

BE IT FURTHER RESOLVED that the Legislation Committee of the North Dakota State Medical Association be instructed to meet at an appropriate time ahead of the Interim Meeting in a legislative year, so that the House of Delegates and the Council may be thoroughly informed prior to the Interim Meeting. A report of the action of this committee meeting should also be forwarded to the constituent medical societies to enable them to take appropriate action.

The resolution from the Stutsman County Medical Society is incorporated in this resolution.

I move the adoption of this Resolution.

Dr. Dunnigan: I second the motion.

Dr. Ulmer: Inasmuch as this in effect amends the By-Laws, this will have to be laid on the table.

Speaker Dahl: We will consider this as a first reading, and if it is passed, it will then be placed on the agenda for final action next year in 1968. All those in favor? All ayes. Motion passed.

RESOLUTION No. 6

WHEREAS, component District Societies should have knowledge beforehand of matters to be considered by the House of Delegates, and

WHEREAS, the individual delegates seldom have sufficient knowledge of such matters in sufficient time to present them to their constituents,

NOW, THEREFORE, BE IT RESOLVED that all resolutions and matters to be discussed, insofar as possible, be submitted to the State Office not later than March 15th of each year so that copies may be sent to the districts by April 1st, and

BE IT FURTHER RESOLVED that matters not so presented require a two-thirds majority vote of the House of Delegates before they can be referred to a committee for action.

This Resolution was adopted.

Speaker Dahl: I do not believe it stipulates in the constitution that a resolution has to be submitted. We are actually setting up a parliamentary procedure in the House itself. It is the chair's opinion that unless there is an appeal, that this is not an amendment to the By-Laws. If the majority approves it, this will be considered the rule.

Dr. Vinje: Anyone can present a resolution. He can read this resolution, but it will take a two-thirds vote of the House before it can be referred to the reference committee of the House of Delegates.

Speaker Dahl: This is so that we can get these resolutions in the Handbook and get them back to the delegates.

All in favor? All ayes. Motion passed.

RESOLUTION No. 7

WHEREAS, the incoming President of the North Dakota State Medical Association has expressed concern about long-range planning for medical facilities, and,

WHEREAS, costs of medical facilities are rapidly increasing, and citizens of our state cannot afford unnecessary duplication of costly facilities and equipment,

NOW, THEREFORE, BE IT RESOLVED that the House of Delegates of the North Dakota State Medical Association directs the Council to implement long-range planning for medical facilities.

This Resolution was adopted.

RESOLUTION No. 8

The following was submitted by the First District Medical Society: "The Society voted to request a resolution to the effect that further preventive measures be recommended in regard to the operation of two-wheel motor vehicles. It was suggested that this would include education of the drivers of such vehicles, perhaps in conjunction with four-wheel driver education in the public schools."

WHEREAS, the request from the First District Medical Society is neither specific nor in resolution form, and

WHEREAS, there will be ample time for the House of Delegates to act on specific recommendations prior to the next meeting of the State Legislature,

NOW, THEREFORE, BE IT RESOLVED that the First District Medical Society is requested to submit a more specific recommendation in resolution form to the next meeting of the House of Delegates.

This Resolution was adopted.

RESOLUTION No. 9

WHEREAS, a need exists for a committee to arbitrate differences, especially as it pertains to fees, between patients and physicians, and especially between third party carriers, and physicians,

NOW, THEREFORE, BE IT RESOLVED that the Committee on Constitution and By-Laws be directed to consider changes in the By-Laws that may be necessary to establish a Committee for Arbitration in each component society, and will outline a specific operating procedure for handling such differences as may arise.

Until such time as the Committee on By-Laws shall take action, the Board of Censors, or local Grievance Committee, (see Chapter XII, Section 5, page 30), and the Mediation Committee (see Chapter XIII, page 32) of the Constitution and By-Laws, shall act in this capacity.

PROCEDURE:

The office of the Executive Secretary shall obtain the names of the Board of Censors, or Grievance Committee, of each society. These names shall be made available to any interested person.

Any complainant shall file a written report with the district Board of Censors, or Grievance Committee. Both parties shall be heard. A formal investigation shall be made by the Board of Censors, or local Grievance Committee who will then arbitrate the differences in a fair and equitable manner. Minutes of this hearing shall be recorded in detail and filed with the district society.

If either complainant or defendant is not satisfied with the judgment, the matter shall then be referred in writing to the Chairman of the Council or to the office of the Executive Secretary who will forward the information to the Chairman of the Council.

The Council shall then act as a Mediation Committee in accordance with Chapter XIII of the Constitution and By-Laws.

A copy of this resolution and a list of each Board of Censors, or local Grievance Committee shall be mailed to each member of the Association.

This resolution and the suggested interim procedures were adopted.

RESOLUTION No. 10

WHEREAS, P. L. 89-97 passed by the 1965 Session of Congress contains many provisions which appear to permit the Department of Health, Education and Welfare to exert unusual interference and control over the manner in which medicine is practiced in this country; and

WHEREAS, Section 1801 of P. L. 89-97 was written to protect the public, the medical profession and hospitals from such interference and control; and

WHEREAS, the intent of Congress expressed in the various committee reports is very clear in that such interference and control is prohibited; and

WHEREAS, Representative Wilbur Mills, Secretary Gardner of HEW, Wilbur Cohen, Arthur Hess and other representatives of this Department have made many public commitments to honor this intent of the Congress; and

WHEREAS, a strict application of Section 1801 of the Law is the only guarantee medicine has against ultimate total federal control of medical practice which was the basis of medicine's objection to this and other socialized medical laws; and

WHEREAS, the AMA House of Delegates in Philadelphia, November-December, 1965, acknowledged the importance of a strict application of Section 1801 P. L. 89-97, and reaffirmed its policy in December, 1966;

NOW, THEREFORE, BE IT RESOLVED that the North Dakota State Medical Association members be alerted to the possibility of violations of this Section of the Act, and

BE IT FURTHER RESOLVED that each district society report any infringement of the Act to the Councillor who will in turn report it to the State Council; and

BE IT FURTHER RESOLVED that a copy of this resolution be forwarded to the Secretary of each district society and to each member of the State Association.

This Resolution was adopted.

RESOLUTION No. 11

WHEREAS, the members of the North Dakota State Medical Association, attending the Eightieth Annual Meeting have thoroughly enjoyed and profited by an excellent scientific program, and

WHEREAS, the Host, the Sixth District Medical Society, and the various chairmen and committeemen have excelled in providing the members of the Association with the graciousness of a fine convention.

NOW, THEREFORE, BE IT RESOLVED that the assembled delegates show their appreciation by a rising vote of thanks.

A rising vote of thanks adopted this Resolution.

RESOLUTION No. 12

WHEREAS, Dr. Glenn W. Toomey, President of the North Dakota State Medical Association for the year 1966-67, did, in the years prior to his term of office, and in the year 1965-66 as President-Elect of this Association, show an intense interest and devotion to the affairs and interests of the Association, and

WHEREAS, with the aid of his experience, knowledge and concern relative to the practice of medicine in North Dakota, and with his dedication to the fulfillment of his office, Dr. Toomey,

did, in such an exemplary manner, lead this Association through the past year,

NOW, THEREFORE, BE IT RESOLVED that the assemhled delegates show their appreciation hy a rising vote of thanks.

A rising vote of thanks adopted this Resolution.

RESOLUTION No. 13

WHEREAS, M. Lyle Limond and his competent staff, Mrs. Margaret Fremming and Mrs. Myrtle Kohler, have served the State Medical Association with diligence and proficiency during the immediate past year, as they have in preceding years

NOW, THEREFORE, BE IT RESOLVED that this House of Delegates formally show its appreciation with the adoption of this Resolution.

This Resolution was adopted.

RESOLUTION No. 14

WHEREAS, the age that the average physician comes in to practice in the State of North Dakota is becoming older, and

WHEREAS, it is becoming evident that fewer physicians are becoming eligible for 50 year honors,

NOW, THEREFORE, BE IT RESOLVED that Article IV, Section 4 of the Constitution and By-Laws be amended to read: "A reputable physician who has been practicing medicine for a period of 40 years after graduation from Medical School shall be eligible for Honorary Membership without payment of dues upon election to Honorary Membership in his local society with the approval of the State Association through its House of Delegates."

This resolution was adopted.

Speaker Dahl advised the members of the House that the resolution was definitely an amendment to the Articles of the Constitution and that this would be considered the first reading. He added that it would be brought up before the next session for final action.

Dr. Vinje moved that the report as a whole be adopted. The motion was seconded by Dr. Hieb and carried.

Dr. E. G. Vinje, *Chairman*
Dr. E. O. Hieb, *Vice-Chairman*
Dr. L. A. Giltner
Dr. J. A. Sandmeyer
Dr. Paul Beithon
Dr. W. C. Hanewald
Dr. Ralph Dunnigan

Dr. Dunnigan moved that the President of the Association appoint a committee to review and up-date the Constitution and By-Laws and that this committee be made up of individuals from a single society and that the members be from the Sixth District Society because of their availability to the Executive Secretary's office in Bismarck. This motion was seconded by Dr. Beithon and passed.

The Chair recognized Dr. R. D. Nierling, Treasurer of the Association, from Jamestown. Dr. Nierling was asked to give his recommendation for dues, both state and national, and to present his report.

Dr. Nierling: If you will turn to page 23 in the Handbook, you can follow this as you go along. There are two things we have to do today, as far as the financial end is concerned. The Treasurer's report here before you, and also determine the dues for the coming year. You will note the balance on hand as of January 1, 1966 was $7,155.68; $46,385.00 were received in dues for the year; showing that we have 465 practicing physicians in the state who pay on an active basis of $100.00 per year. Then you have the interest on U.S. Government bonds listed. The next item is "interest on Time Certificates of Deposit". As you say, the interest on time certificates does not list the number of each C.D., or how many, and the interest is all lumped in the $855.64. Your reference committee number one, mentioned that the C.D. purchased 3-30-66 for $10,000.00 did not show the interest. However, that was lumped in the item of $855.64. The reason is that we have been buying them about this time of year and selling them before the year is up on a 3 month or 6 month basis. This makes a little more interest. We buy a couple a year and sell a couple in a year to obtain this additional interest which would otherwise be left in the checking account.

We have about all the dues in. There is about $4000.00 outstanding. As a consequence we have quite a bit of money in the treasury which we do not need until we get it to Lyle in March, July and November. So, we can keep these C.D.'s from April to November and we can get a certain amount of interest which amounts from $200.00 to $500.00 a year for the Association. That is the reason these C.D.'s are bought and sold. Sometimes you add the interest to the total amount; other times they pay us the interest and put it in the bank account. I would suggest to the treasurer next year, that we list the C.D.'s and the amount of interest obtained. Are there any questions on this statement?

On December 31, 1966 we had a balance of $20,-490.82. You might think we are making a lot of money Actually, Lyle did not need a third of this budget until after the first of the year. We are still functioning within the budget. As you may notice on page 24, U. S. Treasury Bonds are listed. We have had these for quite a while and a couple of years ago at the suggestion of the Council or the House, we did exchange some of them for C.D.'s which gave us more interest. I talked to the banker about exchanging these for C.D.'s at the present time. He looked them over and stated that he did not think it would make much difference. If we cashed them before they matured, we would take a little loss, and we would gain nothing by cashing them into C.D.'s at this time.

If there are no questions on the statement, I would make a motion that this be accepted, with the stipulation and suggestion that the C.D.'s be a little more clarified in the report next year. This report was adopted.

Dr. Nierling: After discussing with Lyle this morning, the expenditure of the budget and the possibility of this year enlarging it, if we do obtain an assistant Executive Secretary, or a Public Relations man, and this was discussed by the Council as it might come to pass this year inasmuch as Lyle is incapacitated. We had Dan Buchanan for a while to help with public relations. I understand from Lyle there is a man who is interested. He is an excellent person; knows the state; has a lot of experience around the state and might be acceptable to the Council for an assistant secretary; helping Lyle and taking over the work which Lyle will have difficulty now to do. If we do that, it might necessitate raising the dues next year, but this year we have enough money. Inasmuch as the House has the prerogative of setting the dues, and we do not know at this time if $100.00 will cover the 1968 expenses, it would be our suggestion to give the Council the prerogative of setting the dues in 1968, at the interim meeting, unless the House will have an interim meeting.

Speaker Dahl: At the second session, the House of Delegates as stated in the By-Laws, fixes the dues for the ensuing year. As I understand it, you would like to have the House give the Council the prerogative of fixing the dues for 1968, at their interim meeting; as we do not know now whether we need to increase the dues or not?

It was moved by Dr. Beithon that the House of Delegates give to the Council the prerogative of setting the dues for the year of 1968 at the Interim Meeting in December. The motion was seconded by Dr. Moses and carried.

The next order of business was the selection of a meet-

ing place for the 1968 annual meeting. Dr. Giltner, on behalf of the Northwest District Society, extended an invitation that it be held in Minot. Seconded by Dr. Huntley and passed.

An invitation was extended by the Third District Society to hold the 1969 meeting in Grand Forks. Seconded by Dr. Powers and carried.

The slate of nominees, as presented by Dr. Foster, Vice-Chairman of the Nominating Committee was voted on singly as follows:

President	V. J. Fischer, Minot
President Elect	C. H. Peters, Bismarck
First Vice-President	R. C. Painter, Grand Forks
Second Vice-President	T. E. Pederson, Jamestown
Speaker of the House	P. O. Dahl, Bismarck
Vice-Speaker of the House	G. L. Countryman, Grafton (Elected for 2 years)
Secretary	W. M. Buckingham
Treasurer	R. D. Nierling, Jamestown
Delegate to the A.M.A.	T. E. Pederson, Jamestown
Alternate Delegate to the A.M.A.	J. J. Moses, Bismarck

Nominated as councillors were Dr. W. R. Fox, Second District; J. M. Van der Linde, Seventh District; Dean Strinden, Eighth District; and W. C. Hanewald, Ninth District.

BOARD OF MEDICAL EXAMINERS

The following 4 names were to be submitted to the governor to fill 3 vacancies on the Board of Medical Examiners; Drs. Keith Vandergon, Grand Forks; P. Roy Gregware, Bismarck; R. M. Fawcett, Devils Lake and G. Christianson, Valley City. There were no additional nominations from the floor. Dr. Sandmeyer moved that the names of these physicians be submitted to the governor. Seconded by Dr. Lommen and passed.

Two names were presented to be submitted to the governor to fill one vacancy on the State Health Council; Drs. L. H. Kermott, Minot, and Paul Beithon, Wahpeton. It was moved by Dr. Helenbolt that approval be given to these two names, seconded by Dr. Countryman and passed.

There being no further business, Speaker Dahl announced that the Council would meet in the same room immediately following the adjournment of the House. Dr. Amidon moved that the meeting be adjourned. Seconded by Dr. Evans and passed. Adjournment was at 4:30 p.m.

SCIENTIFIC PROGRAM

Friday, May 5, 1967

WORLD WAR MEMORIAL BUILDING

Bismarck, North Dakota

9:00 A.M. — Registration

10:30 - 11:15 A.M. — "Present Day Chemotherapy of Tuberculosis," Raymond F. Corpe, M.D., Superintendent, Battey State Hospital, Rome, Georgia. (Sponsored by the North Dakota State Health Department.)

11:15 - 12:00 Noon — "Cardiac Emergencies," Marvin S. Rosenberg, M.D., Assistant Professor of Medicine, University of Illinois College of Medicine. (Supported by a grant from the Merck, Sharp & Dohme — Post-graduate Program.)

NOON RECESS — EXHIBIT VISITATION

2:00 - 2:45 P.M. — "Modern Concepts of Shock and Microcirculation," Louis Orkin, M.D., Chairman, Department of Anesthesiology, Albert Einstein College of Medicine, Bronx, New York. (Sponsored by the North Dakota Heart Association.)

INTERMISSION — EXHIBIT VISITATION

3:15 - 4:30 P.M. — Panel Discussion: "Intensive Care for the Post-operative and Cardiac Patients" and "Treatment of Shock," James F. Houghton, M.D., Moderator, Fargo, North Dakota. M. S. Rosenberg, M.D., Chicago, Illinois; Louis Orkin, M.D., Bronx, New York; Allan Gould, M.D., Rochester, Minnesota.

4:30 - 5:15 P.M. — "Current Treatment of Common Ear Disorders," Neill Goltz, M.D., Department of Otolaryngology, Fargo Clinic, Fargo, N.D.

Saturday, May 6, 1967

9:30 - 10:45 A.M. — "Extended Surgery in Cancer of the Lung," J. Maxwell Chamberlain, M.D., New York, N.Y. (Sponsored by the American Cancer Society, North Dakota Division, Inc.)

10:45 - 12:00 Noon — Panel Discussion: "Therapy of Malignancies," Gale Richardson, M.D., Moderator, Minot, North Dakota.

"Surgical Aspects Related to Chest Disease," John Chamberlain, M.D., New York, N.Y.

"Leukemia, Lymphoma and Related Problems," Leon Jacobson, M.D., Chicago, Ill.

NOON RECESS — EXHIBIT VISITATION

2:00 - 2:45 P.M. — "Anemias and the Control of Red Cell Formation," Leon Jacobson, M.D., Dean, Division of Biological Sciences, University of Chicago, Chicago, Illinois. (Sponsored by Mead Johnson Laboratories.)

INTERMISSION

3:15 - 4:00 P.M. — "Eneuresis," Julian S. Ansell, M.D., School of Medicine, University of Washington, Seattle, Washington.

4:00 - 4:45 P.M. — "Newer Problems in Diagnosis and Treatment of Diabetes Mellitus," Edgar A. Haunz, M.D., Grand Forks Clinic, Grand Forks, N.D.

North Dakota State Medical Association Roster — 1967

MEMBERSHIP BY DISTRICTS

First District

Amidon, Blaine F.	Dakota Clinic, Fargo
Armstrong, William B.	801 6th Avenue N., Fargo
Bacheller, Stephen C.	Enderlin
Bakke, Hans	Lisbon
Barnard, Donald M.	Fargo Clinic, Fargo
Barnes, Michael J.	Hankinson
Bart, Bruce J.	Fargo Clinic, Fargo
Beithon, Elmer J.	Red River Valley Clinic, Wahpeton
Beithon, Paul J.	Red River Valley Clinic, Wahpeton
Beltz, Melvin E.	Wahpeton Clinic, Wahpeton
Bjerke, Leon A.	Red River Valley Clinic, Wahpeton
Borland, Verl G.	Fargo Clinic, Fargo
Burt, Arthur C.	Medical Arts Building, Fargo
Bushell, Robert P.	Fargo Clinic, Fargo
Christoferson, Lee A.	Box 32, Fargo
Christu, Chris M.	Fargo Clinic, Fargo
Darrow, Kent E.	Dakota Clinic, Fargo
DeCesare, F. A.	Dakota Clinic, Fargo
Dodds, G. Alfred	Fargo Clinic, Fargo
Donat, T. L.	408 Medical Arts Building, Fargo
Ehlen, Matt J.	Fargo Clinic, Fargo
Engstrom, Perry H.	Red River Valley Clinic, Wahpeton

Fercho, Calvin K. 304 Medical Arts Building, Fargo
Ferguson, William T., Jr. Dakota Clinic, Fargo
Fortney, Arthur C. Fargo Clinic, Fargo
Foster, George C. 1602 S. 6th Street, Fargo
Gaebe, Robert C. Box 1567, Fargo
Geib, Marvin J. Box 1567, Fargo
Geston, Robert L. 402 Sheyenne St., West Fargo
Gillam, John S. Fargo Clinic, Fargo
Goff, John R. Fargo Clinic, Fargo
Goltz, Neill F. Fargo Clinic, Fargo
Gustafson, M. B. Box 32, Fargo
Hall, G. Howard Fargo Clinic, Fargo
Halliday, D. Ross Fargo Clinic, Fargo
Hamilton, Thomas M. Fargo Clinic, Fargo
Harrington, Ryan B. Box 32, Fargo
Hawn, Hugh W. 624 Gate City Building, Fargo
Haynes, George H. Lisbon
Heilman, Charles O. Fargo Clinic, Fargo
Houghton, James F. Dakota Clinic, Fargo
Hunter, C. M. Medical Arts Building, Fargo
Hunter, G. Wilson Fargo Clinic, Fargo
Irvine, Vincent S. Lidgerwood
Ivers, Robert R. Box 32, Fargo
Jaehning, David G. Hotel Wahpeton, Wahpeton
Jordheim, Robert Fargo Clinic, Fargo
Kana, Dale C. Medical Arts Building, Fargo
Kelly, William A. Medical Arts Building, Fargo
Kohlmeyer, A. C. Box 1567, Fargo
Kulland, R. E. 402 Sheyenne Street, West Fargo
Lamb, Donald L. 204 Medical Arts Building, Fargo
Lancaster, W. E. G.
16333 Redington Drive, Redington, Beach, Fla.
Landa, Marshall Dakota Clinic, Fargo
Lane, Mark H. Box 32, Fargo
Larson, G. Arthur 1538 9th Street South, Fargo
Larson, Leland J. Fargo Clinic, Fargo
Lawrence, Donald H. N. D. State University, Fargo
LeBien, Wayne E. Fargo Clinic, Fargo
LeMar, John D. Fargo Clinic, Fargo
Levine, Leonard Fargo Clinic, Fargo
Lewis, A. K. Lisbon
Lewis, T. H. 302 Black Building, Fargo
Lindsay, Douglas T. Fargo Clinic, Fargo
Lytle, Francis T. Fargo Clinic, Fargo
McMahon, John M. Dakota Clinic, Fargo
Macaulay, W. L. Fargo Clinic, Fargo
Magill, Gordon B. 400 Medical Arts Building, Fargo
Magill, Jane 400 Medical Arts Building, Fargo
Mazur, B. A. Dakota Clinic, Fargo
Melton, Frank M. Dakota Clinic, Fargo
Miller, Herbert H. 509½ Dakota Avenue, Wahpeton
Miller, Joseph W. Dakota Clinic, Fargo
Murray, James B. Dakota Clinic, Fargo
Nagle, Duane W. Fargo Clinic, Fargo
Norum, Henry A. Fargo Clinic, Fargo
Obert, Gerard J. St. Luke's Hospital, Fargo
Ohrt, Harry A. 408 Medical Arts Building, Fargo
O'Hearn, Jerome W. Fargo Clinic, Fargo
Olafson, Richard A. Box 32, Fargo
Olin, Ronald Fargo Clinic, Fargo
Oliver, James 404 Medical Arts Building
Palmer, Wesley J. Lisbon
Poindexter, Marlin H., Jr. Fargo Clinic, Fargo
Poole, Ernest E. Lidgerwood
Pray, Laurence G. Fargo Clinic, Fargo
Rawland, John G. St. John's Hospital, Fargo
Rogers, Robert G. Dakota Clinic, Fargo
Schneider, Joseph F. Medical Arts Building, Fargo
Schutt, Allan J. Fargo Clinic, Fargo
Schutt, Ann H. 702 1st Avenue S., Fargo
Sedlak, Oliver A. 109 Roberts Street, Fargo
Seifert, Richard H. 402 Medical Arts Building, Fargo
Shook, Lester D. Fargo Clinic, Fargo
Spier, J. J. 1720 S. University Avenue, Fargo
Stafne, William A. 2701 12th Street S., Fargo
Story, Robert D. Fargo Clinic, Fargo
Swanson, Joel C. 1220 8th Street S., Fargo
Thompson, George R. Fargo Clinic, Fargo
Thompson, Stanley D. Fargo Clinic, Fargo
Thorfinnson, Hugh D. Medical Arts Building, Fargo
Traynor, Mack V. Fargo Clinic, Fargo
Triggs, Perry O. Fargo Clinic, Fargo
Ulmer, Robert J. Dakota Clinic, Fargo
Veitch, Abner Lisbon
Wall, Wendell H. Wahpeton Clinic, Wahpeton
Wasemiller, E. R. Wahpeton Clinic, Wahpeton
Webster, William O. Fargo Clinic, Fargo
Weible, Ralph D. Dakota Clinic, Fargo
Wenz, E. P. Dakota Clinic, Fargo
Wenzel, James B. Dakota Clinic, Fargo
Weyers, Henry J. 402 Sheyenne Street, West Fargo
Williams, N. S. Dakota Clinic, Fargo
Wiltse, Glenn L. Wahpeton Clinic, Wahpeton
Wold, Lester E. Fargo Clinic
Zauner, Richard J. 300 Medical Arts Building, Fargo

Second District

Cook, Stuart J. Rolette
Corbett, Conner A. Lake Region Clinic, Devils Lake
Coultrip, Raymond L., Jr. McVille (In Service)
Engesather, J. A. D. Lakota
Eylands, J. V. Johnson Clinic, Rugby
Fawcett, John C. Lake Region Clinic, Devils Lake
Fawcett, Robert M. Lake Region Clinic, Devils Lake
Fox, William R. Johnson Clinic, Rugby
Gilbertson, E. V. Lake Region Clinic, Devils Lake
Glasner, Duane D. Rolla Clinic, Rolla
Hillman, R. S. Lyle Johnson Clinic, Rugby
Hilts, G. H. Cando
Johnson, C. G. Rugby
Kihle, Kenneth W. Bottineau Clinic, Bottineau
Lazareck, I. L. 411 Fourth Avenue, Devils Lake
Lind, Jackson W. Bottineau
Lund, A. B. Leeds
McBane, Robert D. Lake Region Clinic, Devils Lake
McIntyre, Donald G. Johnson Clinic, Rugby
Mahoney, James H. 411 Fourth Avenue, Devils Lake
Mari, Justo L. Bottineau
Mattson, Clem J. San Haven
Miller, C. Robert Bottineau
Mimay, Antonio Maddock
Montgomery, Guy E., Jr. Carrington
Neuenschwander, Allen R. Rolla
Overland, A. J. Rolla
Owens, Clarence G. New Rockford
Pine, L. F. Lake Region Clinic, Devils Lake
Rinn, David A. Cando
Sawchuk, John Rolette
Schwinghamer, E. J. New Rockford
Seibel, Glenn W. New Rockford
Terlecki, J. Minnewaukan
Toomey, Glenn W. Lake Region Clinic, Devils Lake
Towarnicky, M. J. Fessenden
Turkula, William Lake Region Clinic, Devils Lake
Voglewede, W. C. Carrington
Wolf, Jerry D. Bottineau

Third District

Barcome, Donald F. U.N.D., Grand Forks
Barric, James G. Park River
Benson, T. Q. 1600 University Avenue, Grand Forks
Benwell, Harry D. 625 Third Street, Grand Forks
Clark, Rodney Grand Forks Clinic, Grand Forks
Clayburgh, Bennie J.
201 South Fourth Street, Grand Forks
Countryman, G. L. 1004 Hill Avenue
Culmer, A. E., Jr.
501 1st National Bank Bldg., Grand Forks
Dailey, Walter C.
Valley Medical Associates, Grand Forks
DeLano, Robert H. Northwood
Doss, R. Douglas 1600 University Avenue, Grand Forks
Eaton, L. P. Grafton Clinic, Grafton
Engelstad, O. D. U.N.D., Grand Forks
Evans, H. W. Grand Forks Clinic, Grand Forks
Flaten, Alfred N. Edinburg
Glaspel, Cyril J. Grafton Clinic, Grafton
Goodall, W. M. Langdon
Gochl, R. O. Grand Forks Clinic, Grand Forks
Graham, John H. 31½ South Third Street, Grand Forks
Grinnell, Ernest L. Grand Forks Clinic, Grand Forks
Hardy, Nigel A. Minto
Harwood, T. H. U.N.D., Grand Forks
Haugen, C. O. Larimore
Haunz, Edgar A. Grand Forks Clinic, Grand Forks
Helenbolt, Kenneth S.
1600 University Avenue, Grand Forks
Helgason, Norman M. Cavalier
Helm, Richard K. 27½ South Third Street, Grand Forks
Hill, Frank A. Grand Forks Clinic, Grand Forks
Isaacson, Peter G. U.N.D., Grand Forks
Johnson, Edward M. Cavalier
Kaluzniak, Nicholas Langdon
Keig, William P., Jr.
1600 University Avenue, Grand Forks
Krahn, Henry Walhalla
Lambie, J. H. Grand Forks Clinic, Grand Forks
Larson, Eric J. Cavalier
Leigh, James A. 111 North 5th Street, Grand Forks
Leigh, Ralph E. 111 North 5th Street, Grand Forks
Leigh, Richard H. 1600 University Avenue, Grand Forks
Lorentson, Carl E. Grand Forks Clinic, Grand Forks
McKay, John F. Grand Forks Clinic, Grand Forks
McLaughlin, Blaine E. U.N.D., Grand Forks
McLeod, John Orthopedic Clinic, Grand Forks
Mahowald, Ralph E. 1400 Grand Avenue, Grand Forks
Mann, Hamish 1600 University Avenue, Grand Forks
Marshall, Robert A. 27 South Third Street, Grand Forks
Meredity, William C. Drayton
Muus, J. M. McVille
Muus, O. Harold 27½ South Third Street, Grand Forks
Nelson, Wallace W. U.N.D., Grand Forks
Nelson, William C. Grand Forks Clinic, Grand Forks
Olson, James R. Grand Forks Clinic, Grand Forks
Painter, Robert C. Grand Forks Clinic, Grand Forks
Panek, A. F. Milton
Pansegrau, Duane F. Grand Forks Clinic, Grand Forks
Peake, Frances M. 14½ South Third Street, Grand Forks
Pettit, Samuel L. Grand Forks Clinic, Grand Forks
Piltingsrud, H. R. Park River
Powers, William T.
Valley Medical Associates, Grand Forks
Prochaska, Leonard J.
517 1st National Bank Bldg., Grand Forks
Ralston, Lloyd S. 33½ South Third Street, Grand Forks
Rand, Charles C. State School, Grafton
Saiki, A. K. U.N.D., Grand Forks
Sande, C. W. Northwood
Sandmeyer, John A. Grand Forks Clinic, Grand Forks
Schafer, Thomas Grand Forks Clinic, Grand Forks
Scheflo, Myron W. 1004 Hill Avenue, Grafton
Silverman, Louis B. Grand Forks Clinic, Grand Forks
Tarpley, Harold I.
Valley Medical Associates, Grand Forks
Teevens, William P. Grafton Clinic, Grafton
Tompkins, C. R. 1004 Hill Avenue, Grafton
Vandergon, Keith G. 1400 Grand Avenue, Grand Forks
Vitums, Arvids State School, Grafton
Warner, Robert C. Grand Forks Clinic, Grand Forks
Wasdahl, Walter U.N.D., Grand Forks
Webber, F. B. Harvey
Wilson, George A. Grand Forks Clinic, Grand Forks
Wilson, Russell H. U.N.D. Medical School, Grand Forks
Woutat, Phillip H. Grand Forks Clinic, Grand Forks
Youngs, Nelson A. Grand Forks Clinic, Grand Forks

Fourth District

Allardice, James G. Stanley
Amstutz, Kenneth N. Northwest Clinic, Minot
Ayash, John J. 123 Second Avenue, S. E., Minot
Barnes, James A. Northwest Clinic, Minot
Bermudez, Enrique Garrison
Blatherwick, Robert Parshall
Boyle, John T. Garrison
Boyum, Lowell E. Harvey
Breslich, Paul J. Northwest Clinic, Minot
Briggs, Brian E. Main Medical Building, Minot
Cameron, Angus L. Northwest Clinic, Minot
Cameron, A. Malcolm Northwest Clinic, Minot
Chanco, Amado, Jr. Powers Lake
Devine, John L., Jr. Great Plains Clinic, Minot
Dormont, Richard E. Northwest Clinic, Minot
Erenfeld, Fred R. 617 North Broadway, Minot
Faulconbridge, A. J. Mohall
Fischer, V. J. Medical Arts Clinic, Minot
Flath, Milford G. Stanley
Flickinger, Dale B. Medical Arts Clinic, Minot
Gammell, Robert T. Kenmare
Garrison, M. W. Garrison Building, Minot
Giltner, Lloyd A. Medical Arts Clinic
Goodman, Robert Powers Lake
Halliday, David J., Jr. Kenmare
Halverson, C. H. 401 South Main, Minot
Hammargren, A. F. Harvey
Hart, George M.
702 Minot Federal Savings & Loan Bldg., Minot
Heidorn, G. H. Main Medical Building, Minot
Hilio, Demosthenes Crosby
Hordinsky, B. Z. Drake
Huntley, Wellington B. 315 South Main, Minot
Hurly, William C. Medical Arts Clinic, Minot
Huss, Bernard, Jr. Medical Arts Clinic, Minot
Jayapathy, B. 123 Second Avenue S.E., Minot
Johnson, O. W. Johnson Clinic, Rugby
Kaljot, Victor Northwest Clinic, Minot
Keck, Stanley W.
702 Minot Federal Savings & Loan Bldg., Minot
Kermott, L. H., Jr. 401 Main Street South, Minot
Kohl, D. L. 123 Second Avenue S. E., Minot
Lampert, M. T. 407 First National Bank Bldg., Minot
Larson, Richard S. Velva
Leonard, Kenneth O. Garrison Clinic, Garrison
Leonard, Kermit Garrison Clinic, Garrison
Liu, Edward Ting Minot EENT Clinic, Minot
London, Carl B. Northwest Clinic, Minot

Naegeli, Frank D. — Northwest Clinic, Minot
Natarajan, Rangasamy — Main Medical Building, Minot
Nazli, Mehmet — 401 South Main, Minot
Odegard, Russell L. — Great Plains Clinic, Minot
Olson, Burton G. — Main Medical Building, Minot
Peterson, Myron D. — U. of Wisconsin Hospital, Madison, Wisc.
Ram, Cecil C. — Medical Arts Clinic, Minot
Richardson, Gale R. — Main Medical Building, Minot
Ringo, G. Roy — 119-A Main Street S., Minot
Rioux, Berchmans — 14-A South Main, Minot
Rowe, Paul H. — Northwest Clinic, Minot
Sahl, Jens, Jr. — Northwest Clinic, Minot
Shea, Samuel E. — Main Medical Building, Minot
Sorenson, Alfred R. — Medical Arts Clinic, Minot
Sorenson, Roger — Medical Arts Clinic, Minot
Stump, Michael M. — Trinity Hospital, Minot
Uthus, O. S. — 21½ 2nd Avenue S. E., Minot
Vandersteen, Paul R. — Mayo Graduate School of Medicine, Rochester, Minn.
Veenbaas, Fred F. — Northwest Clinic, Minot
Wall, Willard W. — Northwest Clinic, Minot
Wallis, Marianne — Main Medical Building, Minot
Westervelt, A. E. — Bowdon
Wilson, Herbert J. — New Town

Fifth District

Berg, Milton O. — Valley City Clinic, Valley City
Christianson, Gunnar — 117 N. W. Third, Valley City
Cody, Gary D. — LaMoure
Craychee, Walter A. — Oakes
Feist, Donald J. — Forman
Goven, John W. — 117 N. W. Third, Valley City
Hockenberry, James A. — Oakes
Jensen, Clayton E. — Valley City Clinic, Valley City
Jensen, Warren R. — 117 N. W. Third, Valley City
Klein, C. J. — 117 N. W. Third, Valley City
Macdonald, Neil A. — 130 Central Avenue S., Valley City

Sixth District

Amos, James R. — N. D. State Health Dept., Bismarck
Arneson, Charles A. — Cowan Building, Bismarck
Arnold, Helmut — Quain & Ramstad Clinic, Bismarck
Baumgartner, Carl J. — Quain & Ramstad Clinic, Bismarck
Berg, Roger M. — Quain & Ramstad Clinic, Bismarck
Bertheau, H. J. — Linton
Boerth, Edwin H. — Workmen's Compensation Bureau, Bismarck
Brink, Norvel O. — Quain & Ramstad Clinic, Bismarck
Buckingham, T. W. — 405½ Broadway, Bismarck
Buckingham, William M. — Capital City Clinic, Bismarck
Candy, Ardis J. — Quain & Ramstad Clinic, Bismarck
Carlsen, Donald A. — 104 2nd Avenue N. W., Mandan
Cartwright, John T. — Missouri Valley Clinic, Bismarck
Chiu, Fang Luke — Turtle Lake
Cleary, Joseph W. — Missouri Valley Clinic, Bismarck
Curiskis, A. A. — Elgin
Dahl, Phillip — Missouri Valley Clinic
Daniolos, D. P. — Quain & Ramstad Clinic, Bismarck
Diven, Wilbur L. — 402½ Main, Bismarck
Dunnigan, Ralph J. — Capital City Clinic, Bismarck
Eriksen, Johan A. — St. Alexius Hospital, Bismarck
Ewert, Artur O. — Beulah
Fardal, Richard W. — Quain & Ramstad Clinic, Bismarck
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Book Reviews . . .

Androgens, Virilization and the Hirsute Female

edited by EUGENE J. SEGRE, M.D., *102 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $6.75*

This book deals with a problem which is common and often perplexing in relation to diagnosis and treatment. The author is an established authority in this field and presents much of his own data and experience in developing a clear and brief essay on the biochemical and clinical abnormalities underlying the virilizing syndromes associated with hirsutism in the female. The first 5 chapters present a discussion of present knowledge in regard to the biochemical disturbances in hirsutism, aimed at the clinician rather than the biochemist. Only a limited basic biochemistry background is needed for understanding. The last 3 chapters are devoted to a brief discussion of the clinical manifestations, diagnosis, and treatment of hirsutism.

In general, the text is well written and clear, but not comprehensive in its presentation. The endocrinologist and investigator in this field may find it too superficial. However, the general internist and general practitioner should gain a clear understanding of the problem without expending a great deal of time and effort.

The text is supplemented by an adequate number of tables and diagrams; however, no pictorial or other illustrations relating to the clinical manifestations are included. The list of literature references is limited but adequate. The book is not a complete discussion of all the virilization syndromes in the female, but deals almost exclusively with the problem of hirsutism.

The book is recommended for the general internist, dematologist, general practitioner and obstetrician, and also for the endocrinologist who wishes an easily digestible, up-to-date review of current knowledge regarding hirsutism in women.

ALVIN L. SCHULTZ, M.D.
Minneapolis

Bronchial Asthma: A Genetic, Population and Psychiatric Study

by DENIS LEIGH, M.D., *and* EDWARD MARLEY, M.D., *192 pp., Pergamon Press, New York City, 1967. $10*

This little book may be of interest to those involved in genetic or psychiatric studies in asthma. The study is presented in a somewhat confusing fashion and unfortunately laeks the clarity of expression for which the British have been noted. The volume describes an investigation by an internist, Denis Leigh, and a psychiatrist, Edward Marley, in which about 1,200 persons were interviewed. The subjects were derived from 2 general practices in the London area. The procedure was conducted entirely by interview of asthmatic subjects matched with controls, as well as their first degree relatives (parents, siblings, and children) and second degree relatives (siblings and grandparents). Conditions under scrutiny, in addition to bronchial asthma, concerned other chest ailments, hay fever, vasomotor rhinitis, urticaria, angioneurotic edema, eczema, peptic ulcer, psoriasis, rheumatoid arthritis, herpes zoster, epilepsy, migraine, and psychiatric illness. Fifty-five asthmatic patients selected from among the London practices were matched with 55 nonasthmatic controls. Also interviewed were over 500 relatives of the asthmatics and slightly less than 500 relatives of controls. Data was also accumulated on 608 deceased relatives, both of asthmatics and controls.

The results of the study indicated that more relatives of asthmatics suffered from asthma than did the relatives of controls. Also, a greater number of asthmatics than controls suffered from chronic respiratory illness. A quarter of the asthmatics experienced hay fever or vasomotor rhinitis whereas only one-tenth of the control population was so affected. Childhood eczema and urticaria were more common in asthmatics than in controls. By the age of 12 more asthmatics than controls suffered from psychiatric disturbances and seemed to suffer more severely. No difference was seen in the prevalence of psychiatric illness among relatives of the asthmatics than among relatives of controls. No significant difference was found in the prevalence of psoriasis, herpes zoster, migraine, rheumatoid arthritis, gastric or duodenal ulcer in the 2 populations. Asthma was found more frequently in the relatives of asthmatics than it was in the relatives of the controls. The suggestion is made that this might be due to dominant type of inheritance with weakened penetrance, but the authors point out that a multifactorial inheritance is also in accord with their results and their study cannot define whether one or many genetic factors are actually involved.

ASHER A. WHITE, M.D.
Minneapolis

Bone Tumors

by DAVID C. DAHLIN, M.D., *2nd ed., 285 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $16*

The general aspects and data of 3,987 cases of histological, verified bone tumors are presented in detail as the author has reviewed the data of the Mayo Clinic. This concise exposition of the problems posed in the diagnosis of tumors and tumor-like processes affecting bones is an excellent reference for physicians and students.

Twenty-six types of primary neoplasms of the bone are considered. The author has reviewed the definition, incidence, sex and age distribution, symptoms and physical findings, roentgenologic features, gross and microscopic pathology, treatment, and prognosis. Excellent pictures of X-rays and pathological specimens are included.

Conditions that commonly simulate primary tumors of the bone are presented briefly, to document some of the differential problems encountered by the pathologist in bone disease. A completely new chapter on odontogenic tumors may be of special interest to general pathologists.

These data of nearly 4,000 bone tumors represent an unusual experience. The book is valuable for the physician and student interested in this aspect of oncology.

B. J. KENNEDY, M.D.
Minneapolis



fall 1967

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this issue: smog, smaze or smust...

Smog, smaze or smust...effects of air pollution on upper respiratory tract

Nathan Flaxman, M.D., Diplomate, American Board of Internal Medicine, Chicago, Illinois

In Los Angeles it is *smog* (smoke and fog). In New York City *smaze* (smoke and haze). In El Paso *smust* (smoke and dust). The original factor was smoke plus such natural phenomena as fog, haze and dust, but air pollution has mushroomed from a smoke problem in our industrial cities into a major economic, esthetic and public health problem that affects practically every American locality and citizen.[1,2] Respiratory disease, of course, is by far the most costly effect of air pollution, for contaminated air can aggravate our illnesses, deplete our strength and shorten our life span.[1]

The greatest problem in dealing with solid wastes is that they are not quickly returned to dust. To aid the decomposing process, the great bulk of such waste is burned, polluting our air in the process.[3] Dr. Jack McKee of the California Institute of Technology[4] has calculated that in Los Angeles County, which has more than six million people, about three pounds of gaseous wastes per person per day (on a dry-weight basis) enter the atmosphere. This is twice as much as solid refuse disposal and six times as much as the contaminants in waste water. It is estimated that in New York City, 730 pounds of pollutants, a little over half the size of a compact two-door sedan of foreign make, is annually thrown into the air for each man, woman and child in the city.[5]

Air pollution is an evident factor, not only in the common cold and upper respiratory disease, but also in chronic bronchitis,[2] pulmonary emphysema,[6] bronchial asthma,[7] pneumonitis and lung cancer.[8] Its effect on the incidence of pulmonary tuberculosis is unproved,[9] although it is conceivable that the presence of various materials polluting the air might do this. A siege of smog in Denver, the "mile high city," in December 1965 was accompanied by respiratory infection that doubled normal absentee rates in schools, factories and city government.[10]

While air pollution is only one factor, it has become important in the causes of most of the afflictions of the respiratory tract. This has been shown not only by the Denver occurrence, but also by detailed study[2] of respiratory illness in a small group of 313 men



from October 1962 to May 1963 when there were 202 episodes involving the upper respiratory tract. The attack rate of illness was related in time to increased concentration of both smoke and sulphur dioxide in the atmosphere of the district in which the men lived.

Other factors often mentioned, include exposure to those who have colds, exposure to extreme changes of temperature, allergy and bacterial infection. However, when low individual resistance due to lack of rest, overwork, fatigue, improper or unbalanced diet, previous illness and emotional stress are included as causes, we enter the realm of somewhat obscure relationships. Much more emphasis can be placed on the role of polluted air.

The symptoms, signs and complications of involvement of the upper respiratory tract, especially the common cold, are the same regardless of the causative factor. Swelling of the lining of the nose, the scratchy dry throat, the discharge from the nose at first watery then thicker, discolored and more tenacious, the eyes tearing, and frequent sneezing are all part of the Number 1 human ailment. Concurrent or residual sinusitis when mucus is trapped there, middle ear involvement due to interference with drainage, laryngitis and bronchitis are complications of the common cold. The primary interference is with a most important function of the nose—the cleansing of foreign matter in the first line of "air defense" to prevent it from entering the breathing tract.

However, the diagnosis and subsequent decision on how to treat the patient so affected rests basically on the relief of symptoms that cause him the misery. The stuffed, runny nose, the clogged ears, and the harsh dry cough—all the symptoms that make common cold sufferers feel miserable and interfere with their sleep—can be alleviated with medications of the oral nasal decongestant/antihistamine combination type. The burning sensation in the throat, soreness of the chest and even chest pain can also be relieved by such medication. Rest in bed if there is fever (but confined to home at least), liberal fluids, uniformly warm surroundings and adequate humidity in the room, are all helpful adjuncts to the medication. Most common cold sufferers recover rapidly and are symptom-free in four to ten days.

Further treatment, altered by the fact that the affliction hangs on for more than the usual duration of the common cold, requires consideration of allergy, which is most frequently the prolonging factor. But air pollution itself may often be the culprit.

(Concluded on following page)

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At the Third National Conference on Air Pollution held recently, it was emphasized that this subject had received more attention in the past four years than in all previous history. Spicer,[11] an active participant at this conference, reiterated that it behooves the practicing physician to be aware of trends in respiratory disease and to accept a major role in community action relating to air pollution and respiratory health. By taking a positive stand physicians have been instrumental in the development of anti-pollution legislation. An outstanding example is Los Angeles where major steps have been taken by abolishing coal burning, and even banishing oil burning, seven months a year. Natural gas must be used instead and it must be used by industry when available. Backyard incinerators have been abolished in favor of landfill disposal, and building incineration ended except for a few expensive smokeless furnaces.[10] Concerted action can be taken against particular industrial nuisances. One company that disregarded complaints discovered its error when thousands of its credit cards were returned by irate customers who decided to patronize competing companies.[12]

Summary. Respiratory disease is the most important and most costly effect of air pollution, whether termed smog, smaze, or smust. Air pollution is an economic, esthetic and public health problem that affects practically every American locality and citizen. New sources of air pollution are invisible and odorless, but the harmful gases and liquid droplets are there. Triggered by sunlight, some of these undergo mid-air chemical changes and the results are even more irritating to the upper respiratory tract. The symptoms, signs and complications, especially of the upper respiratory tract, can be readily aborted by modern medication but may be unduly prolonged by polluted air. In steps taken to prevent this, the practicing physician can take a major role.

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(Advertisement)

News Briefs . . .

North Dakota

DR. D. ROSS HALLIDAY, orthopedic surgeon with the Fargo Clinic for three years, opened an office in the Fargo Medical Arts Building.

* * * *

DR. S. J. THAKOR, part-time director of the North Dakota State Psychiatric Clinic since 1965, joined the department of psychiatry of Quain and Ramstad Clinic in Bismarck.

* * * *

DR. PABLO RAMOS, senior staff physician of Quain and Ramstad Clinic in Bismarck, was certified by the American Board of Radiology.

* * * *

REAPPOINTED to the North Dakota State Board of Medical Examiners was Dr. Keith Vandergon of Grand Forks. New members are Dr. Roy Gregware of Bismarck and Dr. James H. Mahoney of Devils Lake.

* * * *

A ONE-DAY SYMPOSIUM on early diagnosis and treatment of cancer of the gastrointestinal tract will be held Oct. 13 at the University of North Dakota, Grand Forks. Sponsored by the North Dakota Division of the American Cancer Society, North Dakota State Medical Association, and the University of North Dakota Medical College, the program will feature Dr. Julius Wolf, Veterans Administration, Bronx, N.Y.; Dr. A. N. Powers, St. Louis, Mo.; and Dr. Robert Grage, University of Minnesota, Minneapolis.

Minnesota

DR. JOHN W. KARROW, formerly of Mankato, joined the staff of the Granite Medical Center at Granite Falls.

* * * *

DR. A. L. OURADA closed his general practice in Fairmont and Ceylon to start a four-year residency training program in urology at St. Paul–Ramsey Hospital in St. Paul.

* * * *

DR. MICHAEL M. PAPARELLA was named head of the department of otolaryngology at the University of Minnesota. Before coming to Minnesota in April as a professor of otolaryngology, he was assistant professor and director of the otological research laboratory at Ohio State University for three years.

* * * *

DR. JAMES GARBER joined the Olmsted Medical Group after completing graduate studies in internal medicine at Mayo Clinic.

* * * *

DR. WILLIAM J. CARR, physician at the Apache Plaza Medical Center, Minneapolis, spent two weeks as a volunteer in an Evangelical United Brethren Church mission hospital in Beverly, Ky.

* * * *

DR. O. B. FESENMAIER, Brown County coroner for sixteen years, took a leave of absence from his practice in New Ulm and Hanska to do graduate study in Los Angeles, Calif.

(Continued on page 18A)



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NEWS BRIEFS

(Continued from page 17A)

DR. CARL LEMBKE, mayor of Minnesota Lake, completed his fiftieth year as a community physician there.

* * * *

DR. ROBERT RADKE of Montevideo was reelected to active membership in the American Academy of General Practice.

* * * *

THE MAYO CLINIC won six certificates of merit for its four scientific exhibits at the American Medical Association convention at Atlantic City, N.J.

* * * *

DR. W. F. SCHAMBER, formerly of Parkers Prairie, joined the staff of the Veterans Hospital at St. Cloud.

* * * *

DR. GEORGE C. ROTH, chief of staff at St. Paul–Ramsey Hospital, St. Paul, was reelected president of the Ramsey County Tuberculosis and Health Association for 1967-68.

* * * *

DR. ROBERT KIERLAND, head of the dermatology section of Mayo Clinic, Rochester, and chairman of the American Medical Association (AMA) dermatology section, gave a progress report on the Dermatology Foundation at the AMA convention in Atlantic City, N.J. Dr. Kierland is president of the foundation, which is headquartered in New York City.

* * * *

NAMED to American Medical Association (AMA) offices at the annual convention at Atlantic City, N.J., were these Mayo Clinic, Rochester, physicians: Dr. Kinsey Simonton, chairman of the AMA section of laryngology, otology, and rhinology; Dr. Gordon Martin, secretary of the AMA section of physical medicine; and Dr. Lloyd Bartholomew, secretary of the AMA section of gastroenterology.

* * * *

DR. JOHN C. DUFFY, assistant professor in the University of Minnesota child psychiatry division, was named chief consulting psychiatrist for the Minnesota Residential Treatment Center at Circle Pines.

* * * *

DR. CHARLES F. BARBARISI, formerly of Wolfeboro, N.H., joined the staff of the Mankato Clinic, where he will specialize in general surgery.

* * * *

DR. A. M. MCCARTHY of Willmar was elected president of the State Board of Medical Examiners for the coming year.

* * * *

DR. E. P. BRYANT reopened his practice in Ellendale after practicing at the state schools in Faribault.

* * * *

DR. GEORGE W. IBRAHIM, formerly of the Twin Cities, joined the Lenont-Peterson Clinic in Virginia.

* * * *

DR. MIODRAG RISTIC, formerly of Colchester, England, joined the staff at Cambridge State Hospital.

* * * *

DR. JOHN EUSTERMANN of the Mankato Clinic received a grant from the National Institute of Health to study cardiovascular diseases at the University of Miami, Fla.

NEWS BRIEFS

DR. ROALD FUGLESTAD, a native of Moorhead, joined the pathology department of St. Joseph's Hospital at Brainerd.

* * * *

DR. LEWIS E. STRUTHERS, a graduate of the University of Minnesota Medical School, began practice in Parkers Prairie.

* * * *

DR. ROGER A. DREYLING, a native of St. Paul, joined the staff of Paynesville Community Hospital.

* * * *

DR. DONALD MATTSON, formerly medical director and head of the internal medicine department at the Wonju Union Christian Hospital in Korea, joined the staff of Lakeland Medical Center in Willmar as an internist.

* * * *

DR. JOHN KUMAR, formerly of Bombay, India, London, and Edinburgh, Scotland, took up duties as resident physician and surgeon at Divine Redeemer Memorial Hospital in South St. Paul.

* * * *

DR. ANN ARNOLD of Minneapolis was honored by a reception at the Woman's Club upon her retirement from active practice after forty-three years.

* * * *

DRS. HERMAN J. JUST and JOHN W. FODERICK of Hastings moved their offices into the Mississippi Valley Clinic.

* * * *

HONORED for medical assistance to cadets of the Air Force Reserve Officers Training Corps, University of Minnesota, Duluth, were Capt. Roger A. Mattson, director of base medical service, and Capt. Robert M. Levy, flight surgeon, of the Duluth Air Base Flight Surgeon's Office, which was presented a Certificate of Service.

* * * *

DR. DAVID J. LAMUSGA, a native of Browerville, is now associated with the Mora Clinic.

* * * *

NEW ADDITIONS to the staff of the St. Louis Park Medical Center are Dr. Conrad James Wilkowske, formerly an associate consultant in internal medicine at the Mayo Clinic, Rochester, and Dr. Francis J. Everhart, formerly a resident in cardiac pathology at the University of Minnesota.

* * * *

DRS. SHERWOOD YOUNG and STEPHEN HODGSON, both natives of Rochester, were enrolled in the Mayo Graduate School of Medicine.

* * * *

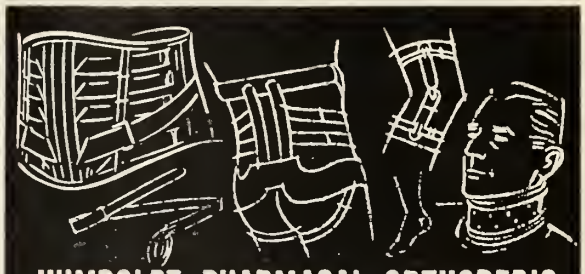
NEWLY APPOINTED to the staff of the Mayo Clinic, Rochester, are Dr. Ralph Nelson, head of the section on nutrition and supervisor of animal research at St. Marys Hospital, and the following consultants: Dr. Raymond Allen, surgical pathology; Dr. Claude Arnaud, biochemical pharmacology; Dr. Frank Leary, urology; Dr. Malcolm Lindsay, Jr., internal medicine; Dr. Walter Lysak, neurology; Dr. Gordon Moore II, psychiatry; Dr. William Taylor, medical statistics, epidemiology, and population genetics; and Drs. Robert Pierre, Mark Schwartz, and Donald Wolochow, clinical pathology. New associate consultants are: Dr. John Clark, dermatology; Dr. Herbert Cooper, pediatrics; Dr. Jack Hall, nephrology; Drs. Elliott Katz and Donald Webb, orthopedic surgery; Dr. John Kelly, neurologic surgery; and

(Continued on page 22A)

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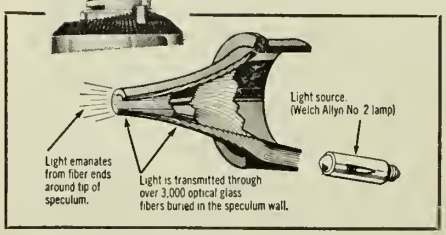
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NEWS BRIEFS

(Continued from page 19A)

Drs. Paul O'Donovan, Roderick Hood, and Richard VanDellen, internal medicine. Appointed chief resident associates in surgery were Drs. Donald Coder and Gerald Woolam.

* * * *

DR. NORBERT O'KEEFE left practice in Spring Valley to take a residency with Quain and Ramstad Clinic in Bismarck, N.D.

* * * *

DR. MALVIN BARER, senior resident in the orthopedic surgery section of the Mayo Clinic, Rochester, spent July and August as a member of a volunteer medical team in Santiago, Dominican Republic, under the Medico Plan, a branch of CARE.

* * * *

DR. GEORGE MYERS, JR., was elected president of the Association of Fellows of Mayo Graduate School of Medicine. He is a resident in urology. Other new officers are Dr. James Hoehn, vice-president; Dr. Gerald Bechamps, secretary; Dr. Thomas Tachovsky, treasurer; and Dr. Franklin Sim, athletic chairman.

* * * *

HOW DRUGS WORK in the human body is the subject of a five-year study for which the University of Minnesota Medical School has received a grant of $373,964 from the National Institute of General Medical Sciences of the Public Health Service.

* * * *

DR. ARNOLD LANDE of the University of Minnesota Medical School was awarded $104,000 by the National Heart Institute to support studies aimed at improving heart-lung machines.

* * * *

DR. JOHN G. MAYNE, instructor in internal medicine at the Mayo Graduate School of Medicine, was appointed to the 14-member National Advisory Committee on Hospital Effectiveness by John W. Gardner, Secretary of Health, Education, and Welfare.

* * * *

DR. THOMAS G. MURN of St. Cloud was elected chief of the St. Cloud Hospital medical staff.

* * * *

DR. GERTRUDE SAXMAN now maintains office hours at the Twin Valley Medical Center.

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NEWS BRIEFS

South Dakota

CERTIFIED in their specialties were two staff members of the Aberdeen Medical Center. Dr. Walter Miller was certified by the specialty board of the American Board of Pediatrics and Dr. Jerome Pucelik by the specialty board of the American Board of Surgery.

* * * *

DR. BERNARD HERZOG, formerly of Sioux Falls, joined the staff of the Milbank Clinic.

* * * *

DR. C. A. STERN discontinued his practice in Sioux Falls to accept a Public Health Service fellowship training grant at the University of California, Berkeley.

* * * *

DR. F. A. MARTIN, who practiced in Faith since 1962, accepted a residency at the Missouri State Hospital at Nevada, Mo.

* * * *

DR. JOHN A. OCHSNER, a native of Wabasha, Minn., joined Drs. James H. Hoskins and John H. Hoskins in the practice of urology at Sioux Falls.

* * * *

ARRIVED to assume duties at Eagle Butte Public Health Hospital were Drs. Alan Crosby, formerly of Ft. Totter, N.D.; Tom Billings, formerly of Kansas City, Mo.; and Larry McEvoy, formerly of Minneapolis, Minn.

* * * *

DR. JOHN FREEMAN, a native of Texas, will join the Hand County Clinic at Miller after a month-long trip with a team of other American doctors to Costa Rica, where they will engage in an extensive missionary program of immunizing the people of that country.

* * * *

DR. JAMES D. COLLINS, a native of Trail City, began full-time practice at the Hoven Clinic and Holy Infant Hospital in Hoven.

* * * *

DR. WILLIAM RENFORTH of Kalamazoo, Mich., was expected to move to Edgemont to begin medical practice.

* * * *

TEN MEDICAL STUDENTS from the University of South Dakota are observing and interviewing 21 South Dakota physicians in a study to find out how doctors get and use information from patients in the diagnosis and treatment of their problems. The project, designed and supervised by the South Dakota Health Research Institute, was granted $42,700 by the Public Health Service.

Deaths . . .

DR. J. A. D. ENGESATHER, 80, Lakota, N.D., died June 24. A member of the Fifty-Year Club of the State Medical Association, he had been active in Lakota-Brocket area civic and medical affairs since the end of World War I, in which he served as a medical officer in France.

Classified Advertisements

RATES: 70 words or less, one time, $3, two consecutive times, $5. Copy must reach us by the 10th of the month preceding publication. REPLIES to advertisements with key numbers should be addressed in care of THE JOURNAL-LANCET, 65th at Valley View Road, Southdale Park, Minneapolis, Minnesota 55424.

GENERAL PRACTITIONER wanted—to join established practitioner. Located 65 miles north of Minneopolis-St. Poul oreo. Populotion over 3,000. Hospital in town. Good schools, churches, and fine fishing ond recreotional area. Contact A. C. Peterson, M.D., 200 E. Forest, Mora, Minnesoto 55051.

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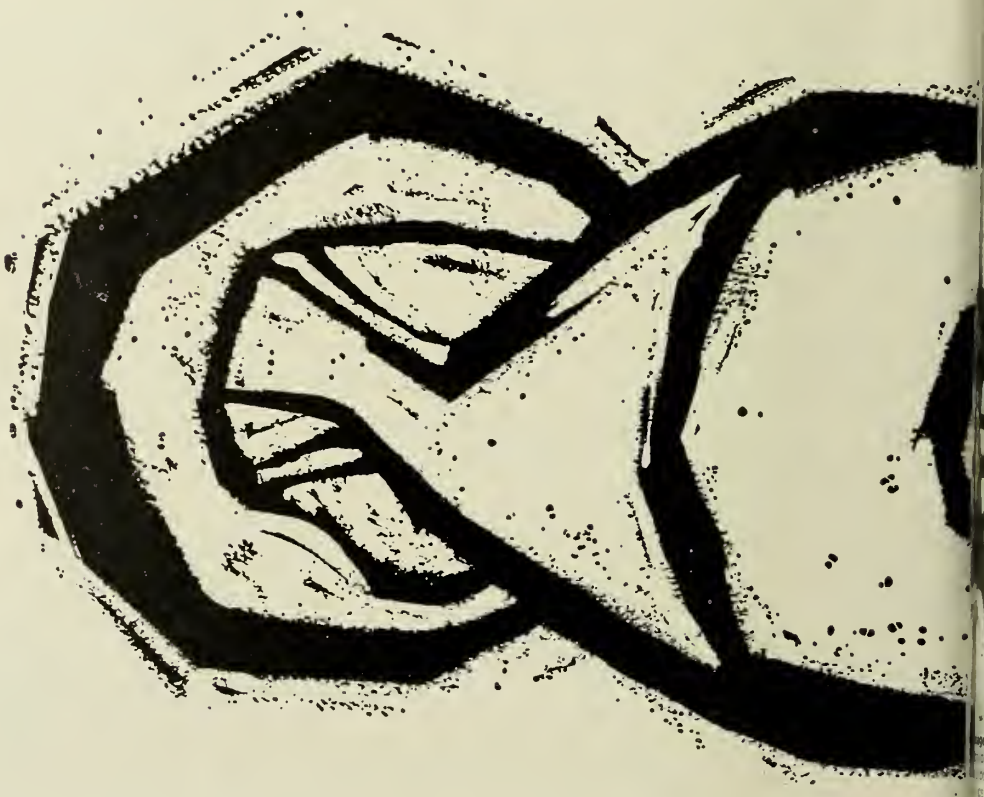
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Contraindications: Local contraindications include obstruction, abscess, perforation, peritonitis, fresh intestinal anastomoses, extensive fistulas and sinus tracts. Tuberculosis (active, latent or questionably healed), ocular herpes simplex, and acute psychosis are usually considered absolute contraindications. Relative contraindications include active peptic ulcer, acute glomerulonephritis, myasthenia gravis, osteoporosis, diverticulitis, thrombophlebitis, psychic disturbances, pregnancy, diabetes, hyperthyroidism, acute coronary disease, hypertension, limited cardiac reserve, and infections. Weigh expected benefits against such risks. If improvement does not occur within 2 or 3 weeks, discontinue use.

Precautions: Administer with caution in severe ulcerative disease because of predisposition to perforation of bowel wall. In advanced chronic disease with loss of mucosa and fibrosis, steroids might hasten deterioration. In severe cases, it is hazardous to delay surgery more than a few days awaiting response to medical treatment. Because of possible adrenal insufficiency, therapy must be withdrawn gradually. If patient is subjected to unusual stress while on steroid therapy or within a year thereafter, adequate supportive measures and increased or reinstated steroids are indicated. For surgery, these measures must be continued throughout pre- and postoperative recovery periods, bea ing in mind possible deleterious effect on fresh intestin anastomoses. Steroids might impair surgical prognosis b increasing hazard of infection. If infection is suspecte administer appropriate antibiotic in large doses. Obser all precautions common to other steroid therapy. Wat for growth suppression in children during long-term us Keep patients under close observation. Discontinue trea ment and institute appropriate measures, if severe rea tions occur. If necessary to use Cortenema in pregnanc examine infants closely for hypoadrenalism; if signs a seen, institute appropriate measures, including steroids.

Adverse Reactions: May produce all side effects that occ with other forms of hydrocortisone, such as moon fac buffalo hump, fluid retention, excessive appetite and weig gain, abnormal fat deposits, mental symptoms, hypert chosis, acne, striae, ecchymosis, increased sweating, pi mentation, dry scaly skin, thinning scalp hair, thrombophl bitis, decreased resistance to infection, negative nitrog balance with delayed bone and wound healing, menstru disorders, neuropathy, peptic ulcer, decreased glucose t erance, hypopotassemia, adrenal insufficiency, necrotizi angiitis, hypertension, pancreatitis and increased int ocular pressure.

In children, suppression of growth may occur. Increas intracranial pressure may occur and possibly account headache, insomnia, and fatigue. Subcapsular catara may result from prolonged usage. Long-term use may res in catabolic effects characterized by negative protein a calcium balance. Osteoporosis, spontaneous fractures a aseptic necrosis of hip and humerus may occur as part catabolic phenomenon.

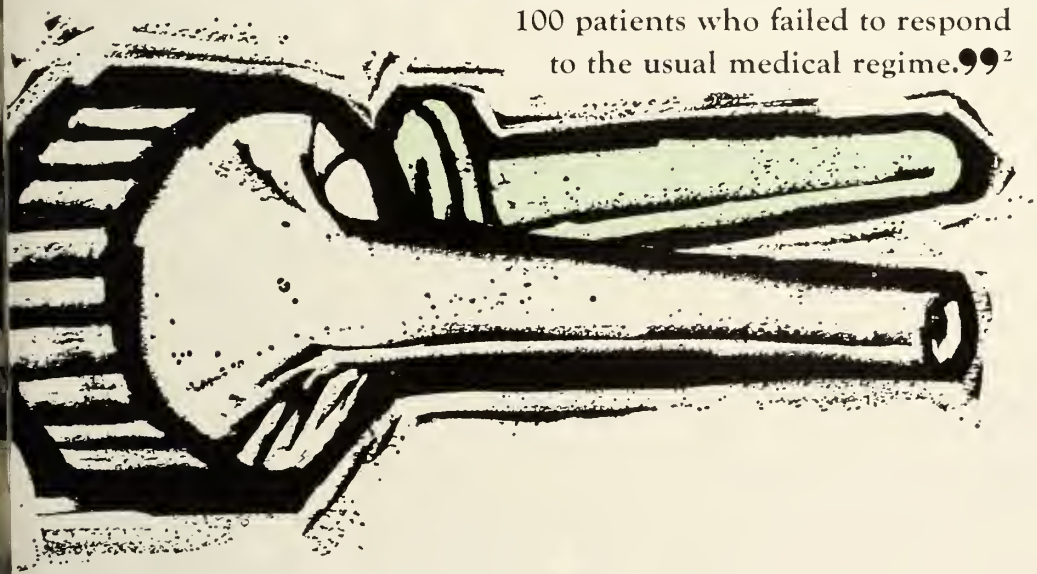
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"safe, efficacious and worthy of trial in most cases of ulcerative colitis"[1]

"...continued good results in more than 100 patients who failed to respond to the usual medical regime."[2]



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n concomitant use of modern supportive measures, such
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ual dose is one Cortenema daily for 2 or 3 weeks and
y second day thereafter, administered intrarectally in
ing before retiring. Every effort should be made to re-
medication for at least one hour, preferably all night.
may be facilitated by prior sedation and/or anti-
heal medication. If improvement does not occur within
or three weeks, discontinue use.

struct patient to lie on left side during instillation.
e bottle vigorously to resuspend hydrocortisone. Ex-
lubricated tip by removal of sheath. *Carefully* insert
to rectum in direction of sacrum. Slowly express con-
by compressing container. After instillation, patient
ld remain on left side at least 30 minutes.

ration of treatment depends on degree of response.
tomatic response usually occurs within 5 to 7 days.
ovement in mucosa, gauged by barium enemas and
idoscopic examinations, may lag somewhat behind
al improvement. Usual duration is two weeks, although
cases require up to 2 or 3 months. Minimal control
mptoms is insufficient basis for prolonged use. If no
al or proctologic response occurs within 2 or 3 weeks,
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nptomatic improvement should not be sole criterion
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t wound healing, Cortenema should not be used in
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1. Sherman, L. F.: The treatment of ulcerative colitis with hydrocortisone alcohol retention enemas. A topical and systemic anti-inflammatory agent, *Minn. Med.* 48:605, May 1965.

2. Sherrnan, L. F., Tenner, R. J. and Schottler, J. L.: Treatment of ulcerative colitis with hydrocortisone alcohol retention enemas: A topical and systemic anti-inflammatory agent. Part II, *Dis. Colon & Rectum* 10:43, January-February 1967.

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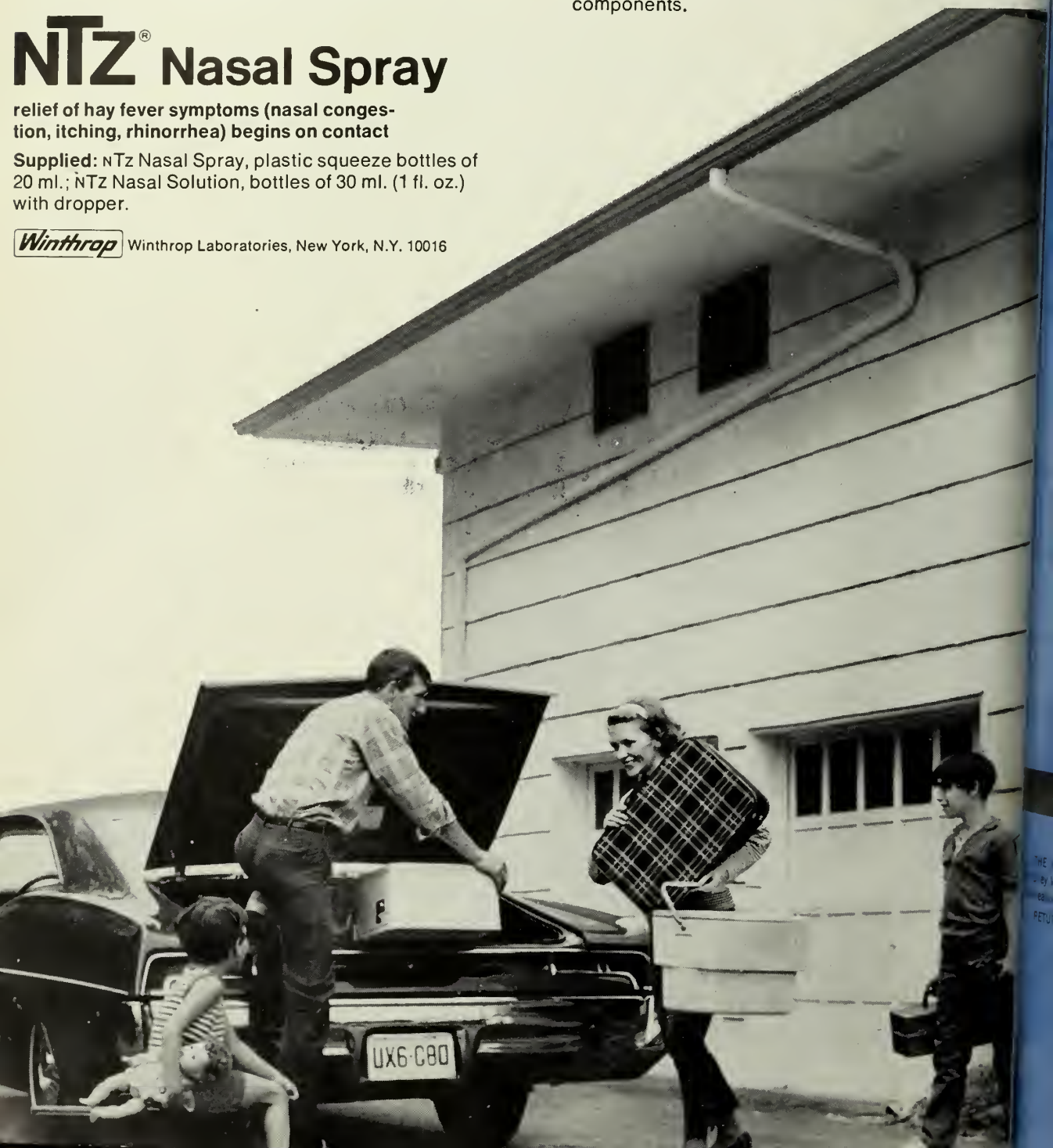
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CODEINE TO SUBDUE COUGH remains the best antitussive and appears to act synergistically with PAPAVERINE in the relief of cold symptoms—and to enhance aspirin analgesia.

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controls diarrhea

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Adults: 2 tsp. 5 times daily (20 mg.) or 2 tablets q.i.d.

*Based on 4 cc. per teaspoonful.

Maintenance dosage may be as low as one-fourth the initial daily dosage.

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Side Effects: Side effects are relatively uncommon but among those reported are gastrointestinal irritation, sedation, dizziness, cutaneous manifestations, restlessness, insomnia, numbness of the extremities, headache, blurring of vision, swelling of the gums, euphoria, depression and general malaise.



1. Barowsky, H., and Schwartz, S. A.: J.A.M.A. *180*:1058-1061 (June 23) 1962. 2. Cayer, D., and Sohmer, M. F.: N. Carolina Med. J. 22:600-604 (Dec.) 1961. 3. Hock, C. W.: J. Med. Ass. Georgia 50:485-488 (Oct.) 1961. 4. Van Derstappen, G., and Vandenbroucke, G.: Med. Klin. 56:962-964 (June 2) 1961. 5. Merlo, M., and Brown, C. H.: Amer. J. Gastroent. 34:625-630 (Dec.) 1960. 6. Weingarten, B.; Weiss, J., and Simon, M.: Amer. J. Gastroent. 35:628-633 (June) 1961.

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Side effects: Occasional drowsiness, blurred vision, cardiac palpitations, flushing, dizziness, nervousness or gastrointestinal upsets.

Precautions: The patient should be advised not to drive a car or operate dangerous machinery if drowsiness occurs. Use with caution in patients with hypertension, heart disease, diabetes or thyrotoxicosis.

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CONTENTS

Atrial Septal Defects 385
C. A. Hamilton, M.D., P. R. Gregware, M.D., P. Ramos, M.D., and R. Fry, R.N.

Diseases of Medical Progress 390
Ronald Olin, M.D.

Acute Myocardial Infarction Complicated by Complete Heart Block: Use of the Transvenous Pacemaker with Observations on a New Demand Pacemaker 393
Waldemar G. Johanson, M.D., and Clarence A. Guenter, M.D.

Psychiatric Hospitalization of Students on a College Campus 397
Carl D. Koutsky, M.D.

Contagious Ecthyma in Man: Report of Two Cases with Laboratory Studies 400
John A. Lambie, M.D., and Arthur A. Gustafson, M.P.H.

Cerebral Abscess and Cyanotic Congenital Heart Disease 403
Richard A. Calkins, M.D., and William E. Bell, M.D.

Ruth E. Boynton Series
Allopurinol in the Treatment of Gout 411
Lydia M. Seebach, M.D.

Patent Ductus Arteriosus: Review of Literature and Experience with Surgical Corrections 415
Col. Daniel C. Campbell, USAF, M.C., Col. Richard H. Hood, Jr., USAF, M.C., and Lt. Col. Byron N. Dooley, USAF, M.C.

The 1967 Influenza B in the USSR 419
Viktor M. Zhdanov, M.D.

Karl W. Anderson, M.D. 421
Earl T. Opstad, M.D.

Woman's Auxiliary to the North Dakota State Medical Association 1967-68 Membership Roster 425

Book Reviews 11A

News Briefs 19A

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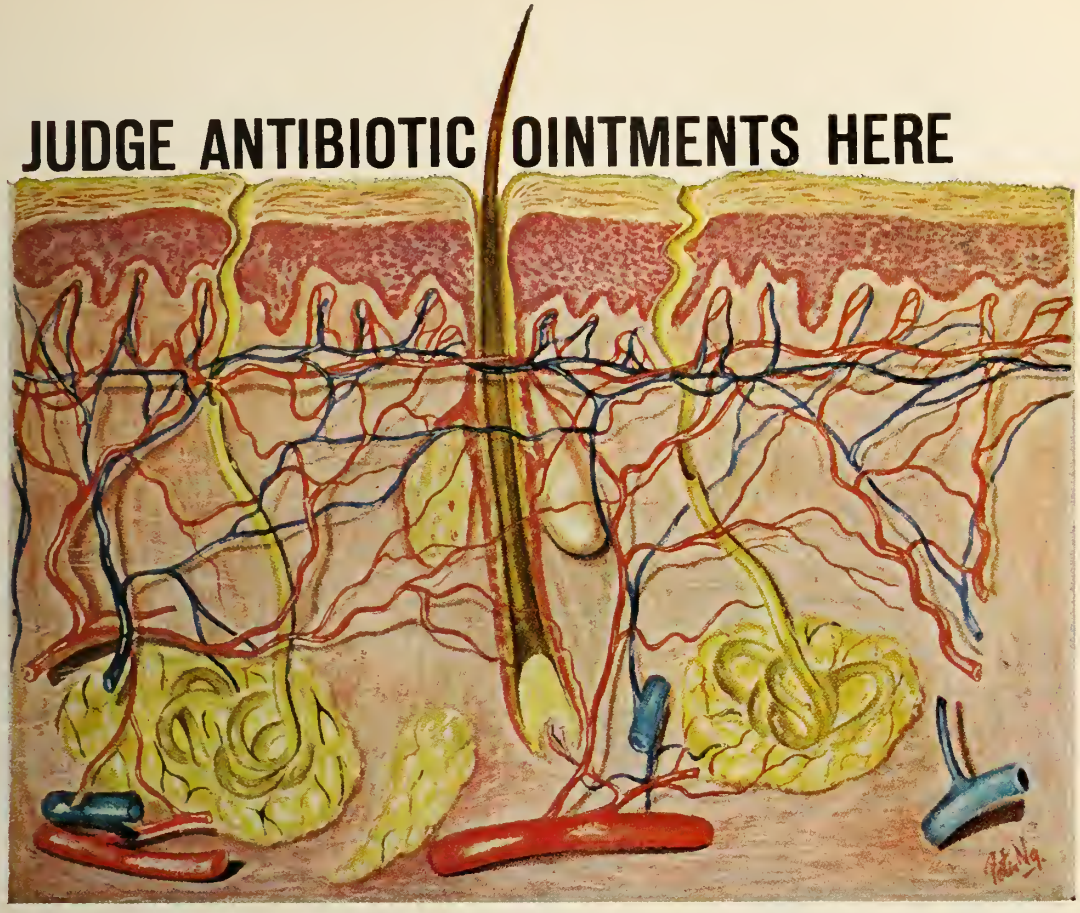
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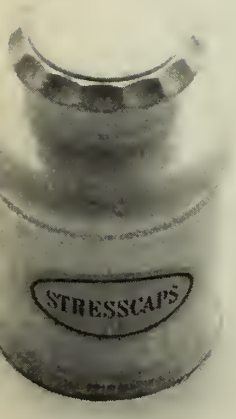
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Atrial Septal Defects

C. A. HAMILTON, M.D., P. R. GREGWARE, M.D., P. RAMOS, M.D., and R. FRY, R.N.

Bismarck

CERTAIN ANOMALIES of the heart are relatively rare. Atrial septal defects associated with tricuspid atresia, dextracardia, scimitar syndrome, and primary hypoplasia of the left ventricle have all been encountered in our practice. However, the most frequent congenital defect which presents in our area is the atrial septal defect.

If large, these defects should be treated surgically but definitive diagnosis must first be made. Since they have unique characteristics, office examination can determine the necessity for more elaborate diagnostic procedures.

Patients with these defects usually come to the physician with a history of fatigue, frequent respiratory infections, failure to gain weight, and palpitations. Office examination will reveal a usual finding of systolic murmur at the upper left sternal border. A more definite lead is the fixed splitting of the second heart tone in the second left intercostal space. Electrocardiograms will reveal incomplete or possibly complete right bundle branch blocks, right axis deviation, right ventricular hypertrophy, and right atrial hypertrophy. These will not all be apparent in the same patient; occasionally the electrocardiographic findings will be normal. However, chest X-rays will reveal increased heart size and increased pulmonary vasculature in most patients. Enlarged pulmonary arteries, ventricles (right or left), and atria (left or right) are frequent.

After a thorough office examination which reveals these leads in conjunction with the presenting history, the patient should be referred for differential diagnosis. Such definitive diagnosis should proceed through multiple still radiographs of the chest and cardiac catheterization with concurrent cineangiocardiography. The defects are then both anatomically and functionally clear and determine whether or not surgical correction is desirable. This study indicates the value of following up these diagnostic leads.

Between 1960 and 1966, 27 definitive diagnoses of septal defects have been made in the heart laboratory of the Bismarck Hospital. Of these, 21 were secundum defects and 6 were primum defects. Fundamentally all septal defects are the same except for their location. Secundum defects, which are the more common, are located in the area of the foramen ovale; primum defects may be considered to be a persistence of the fetal interatrial ostium primum. Diagnostic features of the secundum defects were clear and functional studies served mainly to differentiate the severity of the defect. The symptoms in cases of primum defects are usually more severe than in cases of secundum defects.

Of the signs usually found in office examinations, the findings in the 27 patients were as follows:

Stethoscopic. Abnormal P_2 splits: 16 marked, 5 slight, 2 negative, 4 physiologic. Systolic murmurs were noted in all.

Radiographic. Cardiac enlargement: 11 positive. Increased pulmonary vasculature: 24 positive, 1 borderline, 1 within upper normal limits, 1 negative. Atrial, ventricular, or arterial enlarge-

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ment: left atrium, 2; right atrium, 11; left ventricle, 3; right ventricle, 18; pulmonary artery, 10. Decrease in size of aorta: 14 positive.

Electrocardiographic. Normal, 3. Incomplete right bundle branch block, 11. Right axis deviation, 3. Left axis deviation, 3. Peaked P_2, 1. Right ventricular hypertrophy, 8. Right atrial hypertrophy, 1. P [wave] congenitale, 2.

Each of the patients had some combination of these findings. The table illustrates the diagnostic leads which prompted further detailed heart laboratory studies in the 11 patients who have since come to surgery. (The remaining 16 patients are scheduled for operation, one has already gone to surgery but the reports had not been returned when this was written.) As is

TABLE

Precatheterization findings				*Catheterization findings*				*Operative and postmortem findings or both*	
Patient	*Abnormal P_2 split*	*Chest X-ray*	*Electrocardiogram*	*Number of catheterizations*	*O_2*	*P*	*Shunt*	*Cine grade and findings*	*Size of defect*
1	Yes	↑ Heart ↑ PV ↑ PA ↑ LA and RV	Normal	1	15%	6 mm. Hg	55%	III	5 cm.
2	Slight	↑ Heart ↑ PV ↑ PA and RV	IRBBB	I	10%	8 mm. Hg	56%	II	4 cm.
3	Yes	↑ Heart ↑ PV ↑ RV and LV ↓ Aorta	IRBBB	I	18%	10 mm. Hg	60%	III-IV	3 x 6 cm.
4	Yes	↑ Heart	IRBBB	I	26%	not measured	63.5%	I	2 cm.
5	Yes	↑ PV	RAxD RVH	1	13%	13 to 15 mm. Hg	55%	II	2 to 3 cm.
6	No	↑ PV ↑ RA and RV ↓ Aorta	RVH	I	not calculated (RA%=88)	——	——	II	1.5 cm.
7	Yes	↑ Heart ↑ PA and RV ↑ PV ↓ Aorta	IRBBB	2 preop 1 postop	14%	6 mm. Hg	44%	2 separate ASD each II-III	6 mm. 12 mm.
8	Yes	↑ Heart ↑ PV ↓ Aorta	IRBBB	1 preop 1 postop	15% 0	5 mm. Hg 5 mm. Hg	35-50%	II-III	2 cm.
9	Yes	↑ Heart ↑ PV ↑ RV and PA ↓ Aorta	IRBBB	1 preop 1 postop	12% 0	12 mm. Hg	50%	II-III	Secundum defect confirmed, no size reported
*10	Yes	↑ Heart ↑ PV ↑ RA, RV and LV	PC LAxD	2	13% 17%	—— 2 mm. Hg	50% 50%	II-III Partial anomalous venous return	5 mm.
*11	No	↑ Heart ↑ PV ↑ RA, RV and LV ↓ Aorta	RVH	1	10%	30 mm. Hg	40%	II Complicated by mild pulmonary stenosis	1.5 cm.

Code

↑	Increase in size
↓	Decrease in size or narrowing
*	Primum defects (all others are secundum defects)
IRBBB	Incomplete right bundle branch block
LA	Left atrium
LAxD	Left axis deviation
LV	Left ventricle
O_2	Gradient of oxygen from vena cava to right atrium
P	Gradient of systolic pressure from right ventricle to pulmonary artery
PA	Pulmonary artery
PC	P [wave] congenitale
PV	Pulmonary vasculature
RA	Right atrium
RAxD	Right axis deviation
RV	Right ventricle
RVH	Right ventricular hyperthrophy

Cine grading

I. A whiff of contrast from left to right at atrial septum during atrial systole

II. A definite left to right atrial stream of contrast material during entire cardiac cycle

III. A left to right contrast material shunt sufficient to opacify right atrium and equal to contrasting of left atrium and left ventricle

IV. A left to right contrast shunt of greatest magnitude exceeding contrasting of left ventricle/aorta

obvious from the table, no two patients had exactly the same combination of findings but the clinical finding of an abnormal P_2 splitting should alert the physician to abnormal cardiac findings in chest X-rays and electroeardiograms and any deviation from normal should then suggest immediate referral for finer definition by catheterization and cineradiography.

The table also illustrates the catheterization and cineradiographic findings. These were typical also of the 16 patients who have not yet come to surgery.

The operations were performed at open heart centers in Minneapolis, Portland, Ore., and Rochester, Minn. In each instanee the operative findings confirmed the catheterization and cineradiologie diagnosis.

The 3 case histories further illustrate the developmental program leading from first suspicion to operative correction.

CASE REPORTS

Case 1's history revealed that in 1945, at 35 years of age, a systolie heart murmur was detected. She showed no signs of any eardiae distress and no further investigation was earried out.

In 1956 a heart examination deseribed a systolie murmur in the apieal area and third left intercostal space.

In 1957 she was reported as having had an episode of paroxysmal atrial tachyeardia.

In 1960 she was seen by an internist who described a grade 2 to 3 apieal and 3rd intercostal space systolie murmur. Blood pressure and pulse were normal at that time, but there was questionable liver tenderness. X-ray examination of the ehest showed the heart to be somewhat inereased in size as eompared with previous examinations. An eleetrocardiogram showed an ineomplete right bundle branch bloek. It was felt that the patient was mildly deeompensated and she was digitalized. She was considered to have mitral insufficieney.

This woman was referred to the eardiae eatheterization team for eardiae consultation in November 1964, following an episode of marked palpitation and left bronehopneumonia. Digitalization had been discontinued beeause of a skin rash whieh she thought may have been the result of medication. Her principal complaint was that of rapid heart rate.

Examination revealed a blood pressure of 144/86. The apieal pulse was 160 per minute with a slight irregularity. Following rest, the pulse slowed to 132 per minute. Examination of the lungs was normal. Examination of the heart revealed a gross irregularity with marked pulse deficit. There was a left sternal border systolic murmur, grade 2 intensity. The remainder of the physieal examination was normal. Blood studies showed the hemoglobin was 14.6 gm. and the white eount was 9,000. Urinalysis revealed a 2+ albumin and an oecasional white blood cell. There was mild dermatitis of the lower right leg. X-ray examination of the ehest showed the pulmonary vaseulature slightly inereased. The eardiae size was unehanged from previous examination and was suggestive of a mitral insuffieieney. Cardiac fluoroseopy revealed a hilar danee together with increased pulmonarity vascularity. The left atrial salient was not enlarged. Four days later, examination of the heart revealed P_2 less than A_2, a grade 3 to 4 left sternal border systolic murmur and an audible splitting of the second sound in the left 3rd and 4th interspace. Cardiac catheterization was advised and was carried out on Dee. 1, 1964. Both right and left heart catheterization with oximetrie and manometrie measurements were done as well as cardiac output and cineangioeardiography. There was a significant step-up of the oxygen saturation in the left atrium, suggestive of an atrial septal defect. This was confirmed by cineangioeardiographie studies. Left-sided heart catheterization was performed by the transseptal approach and direct left atrial dye injection was made for cinephotography. Following study of the cinephotography and the oxygen saturation and pressure curves, it was felt that a rather large interatrial septal defect, secundum type, was present. It was ealculated that 55% of the pulmonary blood flow derived from the left to right atrial shunt and surgieal repair of the interatrial defect was recommended.

On Mareh 31, 1965, this patient had surgery performed at a eardiae surgery eenter in Portland, Oregon. At operation a typical foramen ovale type atrial septal defect, approximately 4 or 5 em. in diameter was found. A flap of the foramen ovale was well preserved and it was possible to resuture this flap in such a manner that no prosthetie material was required to elose the defeet. The patient tolerated the operative proeedure well. At time of operation, palpation of the mitral valve revealed it to be completely normal. It was believed that, in this patient, the systolie murmur was a long systolic ejeetion murmur related to the turbulent flow through the right ventriele outflow traet and was widely transmitted over the entire preeordial area.

The patient was again seen by one of our eatheterization team on July 26, 1965. A very faint systolic murmur was present at the left 2nd and 3rd intereostal space. This was ineonstant. X-ray examination of the chest showed the heart size to be smaller than on the preoperative examination and that pulmonary vascularity had returned to normal appearanee. The electroeardiogram eontinued to show ineomplete right bundle branch bloek.

Case 2 was reported to have had a heart murmur since infancy, but no medieal deseription was available. The child was first seen in October 1959, with a history of frequent colds. There was no history of dyspnea, orthopnea, or eyanosis. Examination revealed a thin 6-year-old ehild, weighing 39 lb., with a blood pressure of 100/70. The lungs were clear on examination. Examination of the heart revealed a grade 1 to 2 systolie murmur, maximal in the left 2nd interspace with the patient in a reclining position. No diastolie murmurs were heard. P_2 was split. An electrocardiogram showed an S-1, S-2, S-3 pattern with single r′ in V-1, a large r′ in AVR. X-ray and fluoroseopy of the ehest revealed an inerease in the pulmonary vaseularity. The lung fields were otherwise clear. The heart measurement was at the upper limits of normal aeeording to age. On eardiae fluoroseopy there was a suggestion of right ventrieular enlargement. It was the initial impression that this ehild had an interatrial septal defeet.

The patient was next seen in February 1960. The only complaint at this time was easy fatigability. There was no change in the physieal examination or in X-ray or electrocardiogram findings. Right heart eatheterization was performed on Feb. 9, 1960. During eatheterization the catheter passed from the left antecubital vein to a position felt to represent the right pulmonary vein. This

was confirmed by high oxygen saturation of the blood in this position. The catheter was also readily passed through the atrial septal defect to the left atrium. There was a step-up of oxygen from 67% saturation in the superior vena cava to 76% saturation in the right atrium and right ventricle. The pulmonary artery could not be entered. Diagnosis following cardiac catheterization was interatrial septal defect of the foramen primum type. It was believed at that time that the volume of shunt was not adequate to warrant the rather high risk of surgery.

In January 1962, the child was again seen. Her symptoms included easy fatigue and exertional dyspnea. Lungs were clear on auscultation. Examination of the heart revealed no palpable thrill. On auscultation P-2 was split, but not increased or snapping. There was a grade 2 to 3 systolic murmur, heard best in the 2nd and 3rd intercostal space in the reclining position. No diastolic murmurs were heard. The chest X-ray showed increased pulmonary vascularity and slight cardiomegaly. Electrocardiogram showed left axis deviation with an S-1, S-2, S-3 pattern, r' in V-1, smaller than the initial r. Since a cineangiocardiography was now available to us, the child was scheduled to return for recatheterization.

Cardiac catheterization with cineangiocardiography was performed on Jan. 30, 1962. Cineangiocardiograms were made from both the left ventricle and left atrium. There was no evidence of ventricular septal defect, but a definite atrial septal defect was evident. The patient was referred to an open heart surgical center. Utilizing the heart-lung machine, the patient was operated upon on March 8, 1962. An ostium primum atrial septal defect with complete cleft in the aortic leaflet of the mitral valve was found. An abnormal chordae tendineae was binding down the abnormal leaflet to the ventricular septum. All of these defects were corrected with the use of a teflon prosthesis to close the defect in the ostium primum.

In August 1962, the child was reexamined, at which time a grade 1 systolic murmur was heard best at the apex. The lungs were clear.

On Aug. 21, 1962, recardiac catheterization was carried out. The catheter was readily threaded into the right pulmonary artery. There was no evidence of oxygen step-up. A selective pulmonary artery cineangioeardiogram was done with no evidence of shunt.

The patient has been followed since that time. She continues to do well and, at the time of the last examination on Aug. 25, 1965, she showed normal development. There was, however, a persistent grade 3 systolic murmur at the apex and a slight diastolic blow at the apex was also discernible. Chest X-ray showed no change in the cardiac contour and the pulmonary vascularity was normal. An electrocardiogram persisted in showing incomplete right bundle branch block, with S-1, S-2, S-3 pattern.

Case 3. A heart murmur was first discovered in November 1950, when this child was 3 months of age. Apart from frequent respiratory infections, the patient had normal development.

The patient was first brought to the attention of the cardiac catheterization team in September 1958, at which time examination revealed a well developed 8-year-old female. Cardiac examination revealed a pulse of 102 and regular. There were equal peripheral pulsations. A thrill was palpable in the pulmonic area. The second pulmonic sound was normal in intensity, questionably split. There was a grade 3 systolic murmur in the pulmonic area.

The patient returned for cardiac catheterization in November 1958. At this time X-ray examination of the chest revealed prominence of the pulmonary vascularity with evidence of slight right ventricular enlargement. No posterior chamber enlargement was demonstrated. An electrocardiogram revealed prominent P' waves in leads 1 and 2. There was an incomplete right bundle branch block of the type seen in atrial septal defect of the secundum variety. Catheterization on Nov. 26, 1958, disclosed a gradient of pressure across the pulmonary valve with a step-up of the oxygen saturation in the right atrium, continuing at this same level in the ventricle and pulmonary artery. A diagnosis was made of atrial septal defect, secundum type, with a pulmonary valvular stenosis.

The patient was again seen in December 1960, having had frequent pulmonary infections in the intervening years. This examination again revealed the left upper sternal border systolic murmur, but now there was also a definite splitting of the second pulmonic sound. The electrocardiogram was unchanged from 1958 and showed an incomplete right bundle branch block. The chest X-ray revealed an enlarged right ventricle with prominent pulmonary vascularity. The patient was referred to an open heart surgical center and on Feb. 21, 1961, was operated upon with the use of the heart-lung machine. An atrial septal defect, 2.5 cm. in diameter, of the secundum type was found and closed by direct suturing. The patient had an uneventful postoperative course with no complications. On the day of hospital discharge no cardiac murmurs were heard.

On March 10, 1961, reexamination at our center revealed splitting of P-2 on inspiration and a grade 1 systolic murmur at the left sternal border, compared with the former grade 3 systolic murmur in this area. In August 1961, six months following surgery, the patient returned for followup study and catheterization. The pulse was 80 and regular. The lungs were clear. The right chest incision was well healed. Examination of the heart revealed splitting of P-2 only on inspiration, and, heard best on reclining, grade 1 systolic murmur, over the left sternal border in the 2nd and 3rd intercostal space, but radiating to the apex. The remainder of the physical examination was normal. The electrocardiogram showed little change from earlier tracings. There was a 4 mm. R' compared to a previous 6 to 7 mm. R' in lead V-1 and an incomplete right bundle branch block pattern. In the A-P, oblique and lateral positions, X-ray views of the heart showed little change in the prominence of the pulmonary conus, but somewhat diminished prominence of the other pulmonary vascular markings since films taken before heart surgery.

On August 30, 1961, right heart catheterization and left atrial angiocardiography were performed without sedation. On this occasion no step-up of oxygen saturation was found, but a pressure gradient across the pulmonary valve of 45 mm. Hg was observed, and the pressure pulse contour of the right ventricle was consistent with valvular pulmonic stenosis. Selective left atrial cineangiography showed an intact septum with no left shunt.

In August 1964, there was no change in the physical examination, electrocardiogram, or chest X-ray.

The patient was last seen in August 1966, again with no change in her clinical, electroeardiographic, or X-ray findings. It is proposed to catheterize this patient again at 16 years of age to determine the status of the pulmonary valvular stenosis.

DISCUSSION

The guidelines for diagnosis and operative treatment of atrial septal defects have been discussed by others.[1–5] To effect the best treatment these defects must be reviewed from a broad spectrum of conventional history, physical signs, electrocardiographic and chest X-ray findings, hemodynamic data, and precise exploratory operative techniques.[4,5]

Of the preoperative examinations the most revealing are the combined catheterization-cineangiocardiographic. The relatively simple modalities of hemodynamic studies (right heart catheterization with oximetry, electromanometry, and dye dilution curves) together with phonocardiography, angiocardiography, and cineangiocardiography make it possible for a cardiac team to relate to the open heart team in any other medical unit the details necessary to tailor surgery to the individual case.

The value of a cardiac laboratory in determining the size and position of the defect cannot be overemphasized. However, such a laboratory has equal value in its exclusion capacity. By determining the relative size and severity and excluding those cases which need not come to operation or for whom operation is not an immediate necessity, the regional cardiac laboratory can save the patient the added burden of the time, energy, and expense of trips to the open-heart centers until such time as surgical intervention seems necessary. Of the 191 catheterizations done in our heart laboratory between 1960 and 1966, only the 27 patients discussed in this paper had atrial septal defects; 11 of these were referred to surgery. That the diagnosis made in our regional laboratory has been confirmed by the open-heart surgical teams is indicative of the reliability of the techniques involved. Fewer of our patients have been subjected to risk of surgical closure than those seen in open-heart centers. This, we believe, represents a result of screening a broader spectrum of the size of defects, including a larger percentage of less severe hemodynamic defects which do not require surgical intervention.

SUMMARY

The patient who presents with a history of fatigue, frequent respiratory distress, failure to gain weight, and palpitations should be examined by stethoscopic radiologic and electrocardiographic procedures to determine the existence of the classic symptoms of atrial septal defect.

The classic signs are a combination of an abnormal P_2 splitting, heart enlargement, enlargement of any or all of pulmonary arteries, right or left atrium or ventricle or narrowing of the aorta, together with right bundle branch block and axis deviation or ventricular hypertrophy or both. Not all patients have all these signs but any combination of these should suggest referral to a regional cardiac laboratory for definitive diagnosis.

Definitive diagnosis consists of relatively simple hemodynamic studies involving catheterization (including oximetry, electromanometry, and dye dilution curves), phonocardiography, angiocardiography, and cineangiocardiography. These studies provide screening techniques for determining the existence and relative size and severity of atrial septal defects.

Definitive diagnosis by hemodynamic studies screens out those defects for which surgical treatment is mandatory from those for which surgery is not recommended or should be postponed.

Of 191 patients catheterized in the cardiac laboratory during the years 1960 to 1966, 27 had atrial septal defects. Of these, 11 have already been operated upon in open heart centers. Surgical reports confirmed in each instance the diagnoses made in the cardiac laboratory of the Bismarck Hospital.

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Diseases of Medical Progress

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In the past, a definition of "diseases of medical progress" included iatrogenic disorders induced by autosuggestion based on the physician's examination, mannerisms, and discussion. This, no doubt, led Ben Jonson, a contemporary of Shakespeare, to note in the play *Volpone* that "Physicians are the greatest danger and worst disease to escape." Modern medicine has created an ever-increasing group of artificially induced diseases, so that the definition has been expanded to include disorders resulting from medications and from diagnostic and surgical procedures.[1-3]

Two recent events have focused our attention on the growing problem of iatrogenic diseases. The first was the thalidomide disaster of the early 1960s. The second was a report in 1964 by Schimmel of Johns Hopkins University that 20% of hospitalized patients at teaching institutions experienced a deleterious complication directly related to their medical care.[4] Major complications which were life threatening or a contributing cause of death occurred in 5% of these patients. The lay press played up both these events fully and there was no doubt that the public's confidence in the medical profession was shaken.

The purpose of this paper is to review again the growing problem of iatrogeny. This report cannot review all iatrogenic diseases but only some of the more unusual and interesting ones.

NEUROPSYCHIATRIC IATROGENY

Behavioral disorders can be induced by drugs.[5] This was the subject of an exhibit by the Mayo Clinic at the 1966 meeting of the American Medical Association in Chicago. Grandiose delusions and hyperexcitability can result from ACTH injections or steroids. Impulse rage reactions can occur from stimulants such as anorexiants for obesity. Acute psychosis with agitation can result from meprobamate withdrawal. Psychotic depressions and suicidal tendencies can be induced by the rauwolfia alkaloids in the treatment of hypertension.

Drug-induced neurologic disorders can affect the central and peripheral nervous systems and can also result in myopathies.[6] Convulsive disorders can occur with drug overdosage or in drug withdrawal such as is noted with meprobamate. Cycloserine, chlorprothixene, (Taractan®), and certain phenothiazines have been implicated in some convulsive disorders. Tranylcypromine (Parnate®) and pargyline hydrochloride (Eutonyl®), monoamine-oxidase inhibitors, may precipitate "paradoxical," sudden, severe hypertension with intracranial bleeding when used with amphetamines, other monoamine-oxidase inhibitors, or foods high in tyramine, such as aged cheese. True papilledema with increased intracranial pressure may be induced by steroid therapy. This therapy has been described as a cause of pseudotumor cerebri in children and it is occasionally seen in adults, leading to unnecessary, extensive investigation.

Isoniazid can induce peripheral neuropathies unless pyridoxine is used. Nitrofurantoins can result in peripheral neuropathy in patients with chronic renal failure. Brachial plexus paralysis, with or without associated serum sickness, has been reported following the use of tetanus antitoxin.

Myopathies can be caused by triamcinolone therapy and they seem to be related to the fluorine atom in the alpha-9 position, since they have also been observed with the use of dexamethasone.

METABOLIC IATROGENY

Metabolic disorders can be induced by medications and their causes may often go unrecognized. Thiazide diuretics can induce hyperuricemia with gout and hyperglycemia in diabetics and nondiabetics. Oral hypoglycemic agents such as chlorpropamide can cause prolonged and severe hypoglycemia which may be confused with a stroke in elderly patients, especially if there is no immediate response to intravenous

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dextrose. Gynecomastia may result from therapy with digitalis or spironolactone. Outdated tetracyclines can cause an adult-type, reversible Fanconi syndrome with proteinuria, glycosuria, and aminoaciduria. Busulfan, used in the long-term treatment of chronic myelogenous leukemia or macroglobulinemia of Waldenström, can cause clinical Addison's disease but with normal steroid levels. Iatrogenic Cushing's syndrome with all its sequelae can result from high doses or prolonged use of steroids. Hypercalcemia with a confusing clinical picture of lethargy, weakness, renal insufficiency, and coma can result from vitamin D intoxication, from milk-alkali syndrome, and from estrogen therapy for carcinoma of the breast or prostate with skeletal metastases.

GENETIC IATROGENY

Diseases with a genetically determined diathesis can be uncovered by medications. Hemolytic anemia may be precipitated by many drugs, including primaquine, sulfonamides, nitrofurantoin, and aspirin in patients with a hereditary defect of glucose-6-phosphate dehydrogenase. Polyneuritis resulting from isoniazid therapy seems to be related to a genetically determined rate of acetylation. Patients with atypical cholinesterase may develop prolonged respiratory paralysis during anesthesia because of impaired ability to hydrolyze succinylcholine. Intermittent porphyria may be induced by barbiturates, sulfonamides, and griseofulvin. Systemic lupus erythematosus is probably genetically determined and can be activated by agents of differing potency such as hydralazine, procainamide, isoniazid, and diphenylhydantoin. Some 3 to 12% of cases are drug activated.

GASTROINTESTINAL IATROGENY

Peptic ulceration can occur following the use of steroids, reserpine, phenylbutazone, and salicylates. Pancreatitis has been implicated with the use of ACTH, steroids, and some diuretics, especially chlorothiazide. Hepatic coma can be precipitated by ammonium chloride and chlorothiazide.[7]

Hepatic damage produced by drugs has become an ever-increasing problem and may result from a direct toxic effect or from a hypersensitivity reaction. Drugs which produce direct hepatic toxicity are considered to be protoplasmic poisons. The localization of the lesion depends upon the drug. For example, carbon tetrachloride causes mitochondrial and microsomal injury, whereas the anabolic steroids, for example, norethandrolone (Nilevar®) and fluoxymestrone (Halotestin®) may produce atrophy of the microvilli of the canaliculi. Drug-induced hypersensitivity reactions may result from allergy, with haptene formation evoking antibody that remains attached to the cells. The reaction also may represent individual metabolic susceptibility to specific drug toxicity. A viral hepatitis-like jaundice can result from iproniazid or zoxazolamine. An intrahepatic cholestatic picture characteristic of biliary obstruction can result from chlorpromazine, erythromycin estolate, and a host of other drugs.

Other gastrointestinal diseases associated with medications are pseudomembranous enterocolitis following tetracycline or chloramphenicol therapy, small bowel ulceration induced by enteric-coated potassium chloride, hemorrhagic necrosis of the small bowel during digitalization, mesenteric arteritis caused by norepinephrine, drug-induced toxic megacolon, and malabsorption syndrome like nontropical sprue induced by neomycin.

HEMATOLOGIC IATROGENY

The hematopoietic system is subject to afflictions of every cell line. Leukopenias, thrombocytopenias, aplastic anemia, hemolytic anemia, and megaloblastic anemias have been described.

The phenothiazines are the most common cause of agranulocytosis. Chloramphenicol is the most notorious cause of aplastic anemia. The question whether preloading the patient with phenylalanine will prevent the aplastic anemia is unsettled. Thrombocytopenias can develop from direct toxicity to platelets, as with ristocetin, by depression of platelet production or by an autoimmune phenomenon. With sedormid and quinidine, haptenes are formed so that antibodies develop against the drug-platelet complex. On re-exposure to the drug, thrombocytopenia occurs. Diphenylhydantoin used in the treatment of convulsive disorders can result in megaloblastic anemia as can surgical procedures such as total gastrectomy and small intestinal blind loops or short-circuit procedures.

The widespread use of anticoagulant medication, often without patient cooperation or proper laboratory facilities, has led to a wide variety of iatrogenic problems. In various studies, 2 to 19% of patients on anticoagulants develop hemorrhagic complications. Some of the more unusual manifestations are subdural and epidural hematomas, bilateral adrenal hemorrhage, retroperitoneal hemorrhage, and intramural small bowel hematoma with ileus. Heparin therapy can result in alopecia, osteoporosis, and severe burning and itching of the plantar sur-

faces of the feet and toes (dysesthesia pedis). Unsuspected ingestion of anticoagulants can lead to a hemorrhagic diasthesis.

SURGICAL IATROGENY

Modern advances in surgery have resulted in a multitude of new iatrogenic disorders. Cardiac surgery alone has resulted in several new syndromes including severe, prolonged hemolytic anemia following the insertion of a Starr-Edwards aortic valve and the repair of a septum primum defect with a residual mitral cleft.[8] The mechanism is thought to be mechanical, resulting in red cell trauma. Other postoperative syndromes described include postpericardiotomy syndrome, febrile lymphocyte-splenomegaly syndrome, sequestration syndrome, abnormal bleeding, hypoxic encephalopathy, and psychiatric disturbances. Artificial cardiac pacemakers are widely used and tachycardia and death have resulted from failure in the timing circuit.

The consequences of renal homotransplantation with its prerequisite of immunosuppressive therapy have brought to light several new disorders including fatal pancreatitis and unusual infections such as with cytomegalovirus.

IATROGENIC CANCER

This is perhaps the most dramatic of all iatrogenic diseases. Thorotrast (thorium dioxide), a radioactive contrast medium, was once widely used for cerebral angiography, liver and splenic scans, myelography, and ventriculography and to outline sinus tracts and cavities. Unfortunately, after a latent period of three to thirty-five years, a variety of problems arose including granulomas at the injection site, meningiomas, sarcomas, hemangiosarcomas of the liver, cancer of the maxillary sinus, bile duct carcinoma, and a progressive cauda equina syndrome. The case of Schwartz versus the United States government for carcinoma of the maxillary sinus was settled for $400,000.

Carcinoma of the thyroid can occur following radiation to the neck or mediastinum. The increased mortality rate from leukemia found among radiologists, Hiroshima survivors, and patients with spondylitis point out the danger from radiation exposure. Lymphangiosarcoma of the arm can occur as a sequela of postmastectomy lymphedema. Estrogen therapy for cancer of the prostate has resulted in an increased incidence of cancer of the male breast.

Radioiodine (I^{131}) is now widely used in the treatment of thyrotoxicosis. Pochin[9] reported 18 cases of leukemia in 60,000 patients treated with I^{131} for hyperthyroidism, which was 3 less than the 21 expected to occur by chance in the population. He did not conclude that treatment with I^{131} induces leukemia, but he did not exclude its induction. The radiation exposure to the gonads from I^{131} may have future genetic effects since, in the usual therapeutic dose, it is five times that of a barium enema.

SUMMARY

Iatrogenic disorders have become a major problem and many go unrecognized. The drug-induced disorders are the most common and can result from professional ignorance, drug misuse, allergy, metabolic idiosyncracy, shelf deterioration, and drug abuse. They may result from a single dose or after years of treatment and the reaction may be immediate or delayed.

Sir William Osler said, "One of the first duties of the physician is to educate the masses not to take medicines. Remember how much you do not know. Do not pour strange medicines into your patients." Osler, no doubt, was not advising theapeutic nihilism but rather what Moser has called "therapeutic rationalism." In using a drug such as chloramphenicol, the physician must weigh the risk of the infection to the patient versus the risk of the drug to the patient. Generally speaking, it is true that physicians tend to overprescribe. Patients want medicines and must be educated differently.

Schimmel's rather high and worrisome figure of 20% complications included adverse reactions to digitalis and insulin, both of whose actions have long been familiar to most physicians. Caution is urged in prescribing drugs recently on the market, even if they have been approved by the Food and Drug Administration. It may take years to bring out all the adverse effects.

It goes without saying that caution must be urged in advising drugs for pregnant women and for those who might be pregnant, since the iatrogenic effects are in the first trimester.

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Acute Myocardial Infarction Complicated by Complete Heart Block

Use of the Transvenous Pacemaker with Observations on a New Demand Pacemaker

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COMPLETE HEART BLOCK continues to be a highly fatal complication of acute myocardial infarction. Early experience indicated that this arrhythmia developed in about 5% of all acute infarctions.[1] Several series, based on continuous electrocardiographic monitoring of all patients with acute infarctions, reveal an incidence of about 8% overall[2] and as high as 26% of all inferior infarctions.[3] Paulk and Hurst have reported an incidence of 3.4% in a series of 1,500 admissions for acute myocardial infarction.[4] Prior to the use of transvenous pacing, mortality rates varied from 40 to 100%.[3] The use of transvenous pacing may have brought a decrease in this mortality rate;[4,5] however, critical analysis awaits a wider experience.

Several investigators[4–10] have reported the use of artificial pacing in patients with complete heart block and acute myocardial infarction, emphasizing the immediate improvement in many patients.

The purpose of this report is to present our recent experience with the use of the transvenous catheter pacemaker in dealing with an exceptionally ill group of patients with acute myocardial infarction and complete atrioventricular block. In addition, early experience with a new "demand pacemaker" will be discussed.

METHODS

The patients selected for this study all had electrocardiographic evidence of an acute myocardial infarction or enzyme changes or both characteristic of myocardial infarction (lactic acid dehydrogenase and serum glutamic oxalacetic transaminase).

In all patients the procedure of placement of the catheter was the same. During continuous electrocardiographic monitoring, and external cardiac pacing when required, the right basilic vein was exposed using local anesthesia. The bipolar electrode catheter (United States Catheter Company-C51) was inserted and advanced under fluoroscopic control. It was positioned in the apex of the right ventricle and stability ascertained by putting the patient and the right arm through a range of motion. The threshold for stimulation was determined before the catheter was sutured in place. The position was not considered acceptable if more than 2 milliamperes were required to stimulate the ventricles. The pulse-generator (Medtronics Model No. 5800 or Model No. 5840) was attached and the stimulating rate adjusted to about 68 per minute. In some patients faster rates were used at a later time to suppress ectopic ventricular activity or supraventricular activity.

THE DEMAND PACEMAKER

The demand pacemaker (Medtronics Model No. 5840) used in this study monitors the electrocardiogram at the catheter tip. The electrical impulse is related to the preceding QRS complex by a preset time interval. When the spontaneous rate is slower than the pacemaker rate, the impulse is delivered. When the spontaneous rate is higher than the pacemaker rate, the impulse is inhibited. Thus, the pacemaker may fill

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CASE CHARACTERISTICS, PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND COMPLETE HEART BLOCK

Case No.	Age	Sex	Associated disease	Location of infarction	Day of onset of complete heart block*	Clinical status	Duration of pacing	Course	Outcome
1	65	F	Anemia, aortic stenosis	Inferior	4	Shock, asystole	3 days	Improved, subsequent deterioration	Death, 9th day
2	54	M	Diabetes mellitus	Anterior	2	Shock, asystole	2 days	Marked improvement	Sudden death, 18th day
3	52	F	Obesity	Inferior	1	Shock, asystole	7 hours	Progressive deterioration	Death, 1st day
4	57	F	Diabetes mellitus	Inferior	1	Progressive heart failure	12 days	Marked improvement	Alive and well, sinus rhythm
5	77	F	Diabetes mellitus, pneumonia	Inferior	1	Shock, asystole	8 days	Marked improvement	Alive and well, sinus rhythm
6	81	M	None	Inferior	1	Ventricular fibrillation, shock	Permanent	Gradual improvement	Alive and well, implanted pacemaker
7	55	M	None	Inferior	1	Ventricular fibrillation, shock	5 days	Prolonged hypotension	Alive and well, sinus rhythm
8	70	M	None	Inferior	2	Shock	2 days	Improved, then progressive heart failure	Death, 4th day

*In relation to day of admission

in single beats or pace continuously. If the pacemaker fails to receive a QRS signal, it automatically becomes a fixed-rate pacemaker. The pacemaker will recognize QRS complexes of either polarity greater than 2 millivolts in amplitude and filter out other wave forms such as the P and T waves. (This is made possible by high rejection of low-frequency wave forms, such as the P and T waves, and may be modified by adjustment of the sensitivity control.) The pacemaker may be readily adjusted to function as a demand pacemaker or as a fixed-rate pacemaker.

DESCRIPTION OF CASES

Major features of the case characteristics are summarized in the accompanying table.

Case 1. This 65-year-old woman with aortic stenosis and arteriosclerotic heart disease had been followed in our outpatient clinics for several years. She was admitted with a three-day history of chest pain. The electrocardiogram on admission showed an acute inferior myocardial infarction. She did well until the fourth hospital day when hypotension, diaphoresis, and a pulse of 48 per minute were noted. A nodal rhythm was observed at that time. Intravenous potassium and atropine had no effect. Intravenous isoproterenol increased the ventricular rate to 76 per minute and the blood pressure rose to 120 systolic. When the isoproterenol was slowed, a period of prolonged asystole developed associated with a grand mal seizure. Isoproterenol was then continued until an electrode catheter was inserted and electrical control of her rhythm obtained. Several transitory episodes of asystole occurred during this period. She responded well to the pacemaker and improved for several days. Four days after insertion, while in normal sinus rhythm but with the catheter still in place, progressive congestive failure developed. Two days later she developed ventricular fibrillation and could not be resuscitated. An autopsy was performed with the catheter still in place and revealed the presence of a thrombus in the right axillary vein and thrombi in the right atrium and right femoral vein. A small thrombus was attached to the tip of the catheter. Multiple small pulmonary emboli were present. A massive posterior myocardial infarction was present with old occlusion of the left circumflex coronary artery and recent occlusion of the right coronary artery.

Case 2. This 54-year-old diabetic man entered the hospital with a brief history of chest pain. The admission electrocardiogram showed an acute anterior infarction and an intermittent right bundle-branch block pattern. About thirty hours after admission he had a grand mal seizure with an asystolic pattern on his monitor. Despite intravenous isoproterenol and bicarbonate he had four subsequent episodes of asystole and was very hypotensive. An electrode catheter was inserted without difficulty and pacing instituted. His mental status, which had markedly deteriorated, rapidly improved. He was digitalized and began to diurese. Normal sinus rhythm reappeared six hours after pacing was begun and the pulse generator was turned off. Asystole recurred 12 hours after insertion but responded well when the pacemaker was turned on. He was paced continuously for the next forty-eight hours despite competition with his sinus mechanism. No further episodes of asystole were noted and the

catheter was removed ninety-six hours after insertion. He continued to do well until he died suddenly in bed on the eighteenth hospital day. Normal sinus rhythm was noted on an electrocardiogram taken during a brief agonal stage. Permission for autopsy was refused.

Case 3. This 52-year-old obese, diabetic woman had been in good health until the onset of severe chest pain and dyspnea and an episode of fainting which occurred one hour prior to admission. The admission electrocardiogram revealed the presence of atrial fibrillation and an acute inferior infarction. One hour after admission the ventricular rate slowed from 70 per minute to 40 per minute associated with a fall in blood pressure to 90 systolic. The extremities were cold and clammy. Intravenous isoproterenol increased the rate to 60 per minute but was shortly followed by ventricular fibrillation. A series of direct current countershocks became necessary as fibrillation recurred, each preceded by a period of asystole. An electrode catheter was inserted with one episode of fibrillation occuring during the procedure. Electrical pacing was successful; however, the blood pressure slowly but progressively fell. Efforts to increase the cardiac output were unsuccessful although she continued to respond to the pacemaker electrically. Ventricular fibrillation recurred three hours after insertion of the catheter and resuscitation was unsuccessful. Permission for autopsy was refused.

Case 4. This 57-year-old Negro diabetic woman was admitted because of a two-hour history of marked dyspnea and faintness. The admission electrocardiogram showed an acute inferior myocardial infarction and a 2:1 atrioventricular block. She developed progressive congestive heart failure with periods of complete heart block over the next four days. An electrode catheter was inserted and pacing begun. She diuresed 3,000 ml. of urine in the first twelve hours after the institution of pacing and steadily improved. Sinus rhythm supervened and continuous pacing was discontinued after forty-eight hours but reinstituted intermittently for the next ten days during transient second degree atrioventricular block. The catheter was removed thirteen days after insertion and her recovery was uneventful. She was subsequently discharged from the hospital.

Case 5. This 77-year-old woman presented with a brief history of chest pain. The electrocardiogram showed an acute inferior myocardial infarction. A period of complete atrioventricular block was noted on the day of admission. The following day complete atrioventricular block developed with periods of asystole and grand mal seizures. Isoproterenol was given but asystole recurred. An external pacemaker was utilized while preparations were made for the insertion of an electrode catheter. With the onset of pacing she improved rapidly. Pacing was continued with the demand pacemaker for ten days. No episodes of asystole or syncope occurred during the intermittent demand pacing. She continued to do well and was discharged from the hospital in sinus rhythm.

Case 6. An 81-year-old male had a two-year history of intermittent dyspnea and edema which had been treated with diuretics. Three days prior to admission he developed increasing dyspnea, and on the day of admission he had anterior chest pain, diaphoresis, and marked weakness. At the time of admission he had signs of congestive heart failure. The electrocardiogram revealed atrial fibrillation, an inferior myocardial infarction, and intermittent complete atrioventricular block with a ventricular rate of 44 per minute. Later that day he developed runs of ventricular tachycardia most of which were self-limited but on one occasion required electrical countershock for ventricular fibrillation. An electrode catheter was inserted and pacing instituted. A prompt improvement in mental status and in his heart failure was observed. Three weeks following the insertion of the transvenous electrode, he continued to have complete atrioventricular block, and a permanent pacemaker was implanted.

Case 7. This 55-year-old male had been in good health until one hour prior to admission when he developed substernal chest pain. He was hypotensive and diaphoretic. The electrocardiogram demonstrated sinus bradycardia with a rate of 44, and S-T- and T-wave changes consistent with inferior ischemia. He then developed a nodal rhythm with a pulse rate of 40 per minute. The administration of isoproterenol resulted in sinus rhythm with a ventricular rate of 80 and increased blood pressure. Twelve hours later he developed runs of ventricular tachycardia and on one occasion ventricular fibrillation requiring electrical countershock. Attempts to decrease the rate of administration of isoproterenol resulted in hypotension and complete atrioventricular block. Pacing was instituted via an electrode catheter, utilizing the demand pacemaker. He required intermittent pacing during the next five days and then resumed stable sinus rhythm. Isoproterenol was required to maintain his blood pressure for several days, and the pacemaker was removed on the eighth day. The electrocardiogram demonstrated an inferior myocardial infarction.

Case 8. This 70-year-old male was in good health until four hours prior to admission. He developed epigastric pain and diaphoresis and, on admission, was hypotensive. The electrocardiogram revealed atrial fibrillation with a slow ventricular response and an acute inferior myocardial infarction. Sinus rhythm returned within twenty-four hours of admission and he was doing well until the second hospital day when he developed complete atrioventricular block with a ventricular rate of 40 and hypotension. Pacing was instituted by an electrode catheter, with initial improvement in blood pressure and a decreased central venous pressure. Despite digitalization and intravenous isoproterenol administration, the patient then deteriorated progressively and developed azotemia, metabolic acidosis, and severe hypotension. He died forty-eight hours after the onset of complete atrioventricular block. Autopsy was refused.

DISCUSSION

Transvenous pacing for complete heart block under a variety of situations has been reviewed.[11–13] This technique provides good control of the cardiac rhythm quickly and with little danger to the patient. It also allows digitalization of the failing heart which may otherwise be ill-advised in acute myocardial infarction with heart block.

Our technique, utilizing the basilic vein and lodging the catheter tip in the apex of the right ventricle, has been completely satisfactory. Selection of the basilic vein permits subsequent insertion of a permanent venous pacemaker through the external jugular vein if necessary.[11–13] It may be that our lack of complica-

tions, such as myocardial perforation or catheter dislodgment reportedly due to excessive catheter motion when inserted through an arm vein,[7] is due to the relative immobility of this patient population.

The use of anticoagulants is probably indicated in this situation although not uniformly recommended when transvenous pacemakers are used in other situations.[8,12,13] One of our patients experienced widespread intravascular thrombosis without anticoagulation and the additional factors of shock and congestive heart failure in this group of patients provide sufficient reason for anticoagulation.

The time of removal of the catheter was individualized in every case. Most of the patients demonstrated return of atrioventricular conduction (at least intermittently) within forty-eight to seventy-two hours. When stable sinus rhythm was restored, the pacemaker was usually turned off but the catheter left in place for another twenty-four to forty-eight hours. If further pacing was not required during this period the catheter was removed.

The demand pacemaker was used in two patients and provided optimal control of intermittent atrioventricular block observed in these patients. The potential hazard of ventricular arrhythmias initiated by a paced impulse delivered during the vulnerable period is obviated by this form of pacing.[14,15] Furthermore, the problems of tachycardia due to rapid parasystolic rhythms are avoided. The electrode catheter, electrocardiograph sensing system functioned well through many transitions from sinus rhythm to various degrees of atrioventricular block and vice versa, with no evidence of parasystolic rhythms or asystole.

Other approaches to the problem of acute myocardial infarction with complete heart block have been utilized. A variety of drugs have been recommended including adrenocortical steroids, chlorothiazide, and sympathomimetic agents. All but the latter drugs lack the reliability and immediate onset of action required in some cases but may be of value in a more stable clinical situation. Isoproterenol administered intravenously is of definite value but is not without hazard of inducing ventricular tachyarrhythmias and requires constant monitoring of the infusion. Implantation of a permanent pacemaker has no place in the early management of these patients as at least 80% will revert to their original rhythm within several weeks.[1,16] Several investigators[6,17] have used the transthoracic insertion of a myocardial wire which has been apparently free of complications. This technique offers the advantage of speed of insertion and does not require fluoroscopic control, but it requires the blind insertion of a needle guide into the myocardium. An adaptation of this technique proposed by Roe[18] utilized a hooked wire which requires a thoracotomy for removal and is not applicable to this situation.

It is not established that the high mortality among patients with acute myocardial infarction and complete heart block is due to the rhythm disturbance alone,[6,19,20] although some authors[5,7–10] have felt that the abnormal rhythm poses the major threat to life. The high mortality in our cases, despite successful management of the heart block, would indicate that factors other than the rhythm disturbance alone are significant.

It was clear that death was imminent in the majority of our cases as they developed recurrent episodes of cardiac arrest, ventricular fibrillation, syncope, and hypotension. Of the 4 patients who died, 2 developed progressive heart failure despite effective electrical pacing, 1 had documented pulmonary emboli, and 1 died suddenly on the eighteenth day of unknown cause (sinus rhythm was documented on an agonal electrocardiogram).

The mortality rate of 50% in our patients was somewhat higher than in the series reported by Paulk and Hurst[4] and Bruce and associates.[5] This may reflect a patient population with more severe disease, since all but one of our patients had syncopal episodes or profound shock prior to the institution of pacing.

SUMMARY

Eight cases of acute myocardial infarction and complete heart block treated with transvenous pacemakers are presented. Seven of the patients responded dramatically to pacing with rapid improvement of a desperate clinical situation. One of these subsequently developed progressive heart failure and died. Two others died later in their course following the restoration of normal sinus rhythm. This emphasizes that factors other than the arrhythmia alone play an important role in the eventual outcome of such patients.

The use of a demand pacemaker in two of the patients permitted optimal control of the ventricular rate during the recovery of atrioventricular conduction.

Transvenous pacing provides an effective means of coping with the problems of syncope, shock, and congestive failure secondary to complete heart block associated with acute myocardial infarction.

(References continued on page 414)

Psychiatric Hospitalization of Students on a College Campus

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Behavior on a college campus is often viewed critically by the average noncollege citizen, and such things as panty raids and reports of other isolated but suggestive and sensational activities reflect readiness of the "older generation" to believe that the major part of social activity on campus must somehow be pathologically distorted. Psychiatric hospital units are often the reception centers for the most disturbed behavior of an emergency nature; therefore, to find out more of the truth about students needing rapid psychiatric hospitalization in a university community, we employed an IBM registry system recently set up in our inpatient psychiatric service and tabulated data about hospitalization of our students over the past two years. These findings were then compared with data on patients in other psychiatric situations.

MATERIAL AND METHODS

Ninety-four patients who were registered as university students were admitted to our service between January 1963 and January 1965. This was about 10% of the total number of admissions (977) to our inpatient unit, which is a 68-bed inpatient service for adults. All but 5 of the students admitted were citizens of the United States, and the age range of the group was from 17 to 45 years, with 83% falling between the ages of 19 and 26. Sex distribution favored females 54 to 40. These students had been born in 12 states of the United States and in 5 foreign countries, and 87% of them were Caucasian. Sixty-one came from the state of Minnesota, with 53 coming either from the county in which the University of Minnesota is situated or from the neighboring county; however, there were some Minnesota-born students from 22 of the 87 counties that make up the state. Thirteen students were married, only 2 had a record of divorce, and 1 student was separated from his spouse.

In Table 1, the student case sample is categorized into 4 diagnostic groups and compared with those of a routinely admitted group of 86 patients. The student group showed a considerably greater incidence of schizophrenic reactions and personality disorders and a lower incidence of psychoneurotic reactions than did the comparison group. This observation is evidence that psychoneurotic reactions and personality disorders are ordinarily handled on an outpatient basis through our Student Health Service Outpatient Center and that patients with neuroses are usually not admitted to hospital, even though they sometimes have quite severe reactions. The one exception is in cases of depressive syndromes, most often classified in psychoneurotic or personality disorder groups, although seriously depressed. The only diagnosis of physical illness in the student group was made on a student who became intoxicated as a result of chronically abusing sedative drugs.

The students in this sample were referred to the hospital from several sources: 58 came directly through the Student Health Service, 18 were being seen by private psychiatrists in the surrounding community, and the remaining 18 were referred through various facilities, including nonpsychiatric private physicians, courts, state mental health centers, and state hospitals. Sixty-five of the student-patients were experiencing their first admission to the hospital, 20 were in the hospital for their second admission, and 9 for their third or fourth admission. Thus, 31% had one or more previous hospitalizations for psychiatric purposes. The great majority of students (74) were in their first, second, or third year of college; 20 were in their fourth year or were in a professional or graduate status.

The treatment available for the students is the same for patients routinely admitted to the psychiatric unit. Primary medical responsibility is

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assumed by the full-time psychiatric staff who delegates this responsibility to a psychiatric resident and a junior clerk. However, the primary therapist varies, depending on special treatment needs and teaching or research, or both, criteria. As a result of these considerations, 76 of the 94 students were seen primarily by resident psychiatrists, 16 by staff psychiatrists, and 2 by senior medical students who were taking specialized work in the department of psychiatry.

Once it is determined that definitive inpatient treatment will be conducted at the University Hospitals, a treatment program specifically designed for the particular case is started. This program is based on the individual psychotherapeutic relationship that is determined to be the most advantageous by the team in charge of the treatment. In addition to employing traditional psychotherapy and psychopharmacologic therapy, other treatment procedures may be employed. For example, 38 student-patients had extensive psychological testing, 6 had electroshock therapy, 40 were included in a group therapy program in the hospital, 8 received vocational counseling, and 9 received rather extensive social service work from the social service department. The length of hospitalization ranged from one day to two hundred forty-three days, with 50% of the student-patients staying less than forty days and 85% less than one hundred days.

RESULTS

The results of treatment at discharge were as follows: 31 student-patients had regained their previous state of health, 21 had experienced moderate improvement, 9 had experienced mild improvement, 4 were minimally improved, 12 remained the same, and 7 were seen only for evaluation while in the hospital. Eighty student-patients were discharged from the hospital on a regular discharge basis, 4 were committed to state hospitals for further care, 7 left the hospital against medical advice, 2 eloped, and 1 discharge was classified as other. Of 94 student-patient discharges, 80 left our hospital without need for commitment, while 4 had to be legally committed to state hospitals. In the routine admission group used for comparison (Table 1), 79 of the 86 patients were discharged, and 7 were committed. This limited view does not reflect better results in college student-patients.

TABLE 1

DIAGNOSTIC CATEGORIES OF ADMISSIONS TO PSYCHIATRIC SERVICE

	Routine (15 to 65 years)	*Student (17 to 45 years)*
Schizophrenic reactions	36 (42%)	55 (58%)
Personality disorders	9 (10%)	18 (20%)
Psychoneurotic reactions	25 (30%)	17 (18%)
Psychotic depressive syndromes	7 (8%)	2 (2%)
Other	9 (10%)	2 (2%)
Total:	86	94

As is our routine procedure with all patients, the student-patients were discharged from the hospital with planned continued care, mostly in the university outpaint department; 17 were returned to the Student Health Service, while 12 were referred to private psychiatrists.

A look at related data reveals some significant findings. The average number of students eligible for health service care annually was about 32,000 (63,862 students were eligible during the two-year period of the study). Using readily available data[1,2] and Gregory's definition of rate as "the number of occurrences at or within a given time, divided by the total number of subjects at risk of this occurrence,"[3] the following interesting comparisons can be made. The ages of the students in our sample ranged from 17 to 45 years. Sixty-five of our patients were experiencing their first psychiatric hospitalization for an admission rate of 1.01 per 1,000 annually. Population statistics for the state of Minnesota[1] indicate that between the ages of 25 and 44 (the most similar age group readily available for comparison) the population has remained relatively stable from 1940 to 1960. There were 406,733 in this age group in 1940, 413,312 in 1950, and 404,701 in 1960.[1] The Minnesota State Mental Hospitals Statistical Report[2] for 1963-64 reported 615 first admissions in Minnesota state hospitals in the 25 to 44 age group, which gives a rate of 1.51 per 1,000 population for first admissions for that age group. Comparatively, rate of admission to the hospital at the university is considerably less than the rate of admission of the general population to state hospitals.

DISCUSSION

Attempts to explain the difference in admission rates in the university population and in the state hospital population lead to two speculations. First, the difference may be even greater because many patients are treated in psychiatric units of general hospitals and are not recorded in the state hospital admission rate. This is probably balanced by the student population who seek hospital care away from the campus where they are known, since they do not want a psychiatric admission on their student record. Secondly, analysis of occupational and eduea-

tional levels, using the Hollingshead Two Factor Index of Social Position[4] as a guide, showed marked differences in the level of the household head's education and occupation in the student groups compared with a nonstudent group selected at random in a comparable age (19 to 27) range and admitted to the same psychiatric unit (Table 2). Perhaps the most significant difference between the groups is seen by comparing the background of the patients involved.

To seek an explanation for those admissions that did occur in students, it was postulated that specific stresses might be peculiar to the process of obtaining a college education. An attempt was made to analyze the frequency of admissions with reference to final examinations and end-of-quarter failures. Over the two-year period, there was at least one admission every month, with a peak number of admissions occurring in May and October of 1963 and in February and March of 1964. Examination of class and final examination schedules revealed no correlation between any particular time when scholastic stress was likely to occur and an increase in the hospitalization of students for psychiatric reasons. Because the lapse of time between a precipitating stress and the resulting psychiatric crisis cannot be specified, it is impossible to say that there is not stress from this type of activity. However, from the data collected, scholastic stress cannot be pinpointed as the major precipitating stress leading to psychiatric hospitalization of students. The whole question of scholastic stress and its relation to hospitalization needs further study.

TABLE 2

EDUCATIONAL AND OCCUPATIONAL FACTORS OF HOUSEHOLD HEADS[4]

Educational level	*Student*	*Nonstudent*
More than four years of college	35	11
4 years of college	10	8
1 to 3 years of college	14	12
High school graduate	19	34
9th to 11th grades	5	16
8th grade	9	14
7th grade or less	0	6
Unknown	2	0
	94	101

Occupational level	*Class*	*Student*	*Nonstudent*
Executive and major professionals	I	28	11
Business managers	II	21	5
Administrative personnel	III	18	11
Clerical and sales workers	IV	9	15
Skilled manual employees	V	5	20
Semiskilled employees	VI	6	15
Unskilled employees	VII	4	24
Student supporting self		2	0
Unknown		1	0
		94	101

COMMENTS

Almost 80% of the students became psychiatrically incapacitated before their fourth college year. Thus, if a patient is to be rehabilitated and returned to the university community in as normal a capacity as possible, a student health service should provide continued treatment on an outpatient basis for a majority of such patients.

Student health service personnel might wonder if the university is being used as a rehabilitation center for psychiatric patients. However, if we assume that the rate of admission for students is less than it is for the general population, and this is so in the present study, we would have to say that the university is probably not being used as a rehabilitation center any more than any other industry, training institution, or labor market at this time. Rather, the university student group as a whole tends to present a better mental picture than comparable populations in nearly the same age groups, although once psychiatrically ill, the treatment results are similar to nonstudent groups. The finding that students' families had better educational and occupational backgrounds is further confirmation that "success breeds success" and is evidence tending to support programs expanding educational opportunities for more young people. It should also be noted that only 1 of the 94 students diagnosed was involved with drugs. The popular fear of LSD, marijuana, and "Beatniks" is not reflected in acute psychiatric hospitalization. If this can be considered a significant scale from which to judge, the results are reassuring.

SUMMARY

Data collected from an IBM registry on 94 students admitted to a hospital for psychiatric care, when compared with data for a similar group of nonstudent patients, show a better mental health picture for the student group.

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Contagious Ecthyma in Man

Report of Two Cases with Laboratory Studies

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ECTHYMA CONTAGIOSUM is a rarely reported disease of man. It is primarily a disease of young sheep and goats and is known to sheep men as sore mouth disease. Viral in etiology, the disease causes high morbidity and some mortality in sheep. Infection in man is self-limiting, producing skin lesions which are often painful but which heal spontaneously.

Recently, two farmers with the disease were seen and treated. Their case reports and laboratory studies are presented together with a review of the literature.

CASE REPORTS

Case 1. A 40-year-old white male was seen in the Grand Forks Clinic on Dec. 23, 1963, complaining of painful lesions of the fingers present for several days. He had administered oral medications to a flock of sheep on Dec. 4. This necessitated forcibly opening sheeps' mouths and inserting capsules by hand. As a result, numerous small abrasions of the patient's hands occurred from scratches by the teeth of sheep during treatment. Some of the sheep were known to have had sore mouth disease.

Examination revealed skin lesions of the right second and left third fingers. The lesion of the right second finger had a pustular-appearing center surrounded by an outer raised violaceous ring. The lesion of the left third finger was a fluid-filled vesicle surrounded by an area of erythema. Tender, soft, enlarged left epitrochlear and left axillary lymph nodes were palpable. The remainder of the physical examination was unremarkable.

Laboratory studies disclosed the following values: hemoglobin, 16 gm.; white blood cell count (WBC), 8,250; polymorphonuclear neutrophil leukocytes (P.M.N.), 64%; lymphocytes, 33%; and monocytes, 3%. A routine urinalysis and chest X-ray were normal.

The patient was treated with tetanus toxoid (0.5 cc. intramuscularly), penicillin, and tetracycline. The pustular-appearing lesion was opened and found to contain no pus or fluid. Culture from the opened lesion showed coagulase-positive staphylococcus felt to be a contaminant. Because of continued pain and enlargement of the lesions, debridement was carried out three days later in the hospital.

Specimens were sent to the public health laboratory for further study, which included clear straw-colored vesicular fluid, skin scrapings, small pieces of excised skin, and venous blood. Vesicular fluid was diluted with equal amounts of phospate-buffered broth containing 5% calf serum. Cultures on blood agar and in thioglycolate broth and trypticase soy broth were negative. Fungus cultures on Sabouraud's dextrose agar and mycophil agar were negative. The blood clot was removed aseptically from the serum, macerated, and cultured for fourteen days in trypticase soy broth with negative results. Agglutination tests against typhoid H, typhoid O, paratyphoid B, brucellosis, tularemia, Proteus OX-19, Proteus OX-2, and Proteus OX-K antigens were negative. A venereal disease research laboratory (VDRL) test for syphilis was nonreactive. The remainder of the vesicular fluid and skin scrapings were frozen at –20° C. for future use.

Following debridement, the lesions were treated with warm saline soaks and Neo-polycin® topical ointment. Rapid healing occurred, and the patient was discharged five days later.

Case 2. A 31-year-old white male was seen in the Grand Forks Clinic on Dec. 6, 1963, with painful lesions of the fingers. He was the employer of Case 1 and also had been scratched by sharp teeth while giving the sheep oral medications two days earlier.

Examination revealed papulovesicular lesions with surrounding areas of erythema of the right second and third fingers. An enlarged axillary lymph node was present. The remainder of the physical examination was normal.

Laboratory studies disclosed the following values: hemoglobin, 13.9 gm.; WBC, 5,800; P.M.N., 49%; stab cells, 16%; lymphocytes, 28%; monocytes, 3%; and eosinophils, 4%. Routine urinalysis was normal. Bacteriological and fungal studies at the public health laboratory were essentially negative, as with Case 1.

The patient was given tetanus toxid (0.5 cc.) and oxytetracycline (Terramycin®). Debridement was done in the hospital on Dec. 19. Penicillin was administered, and warm, moist saline soaks were applied to the lesions following debridement. The lesions healed, and the patient was discharged.

Epidemiological study of the 2 cases revealed that the individuals were feeding approximately

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1,000 western range (Montana) sheep in preparation for marketing. Questioning of the patients revealed that many of the sheep had crusted lesions on the nose and lips. With this clue and careful perusal of the literature concerning pox-like diseases of sheep, contagious ecthyma was considered a strong possibility. A local veterinarian examined the animals and felt the lesions were classical for contagious ecthyma.

METHODS AND MATERIALS

A portion of the vesicular fluid and skin scrapings from Case 2 was sent to the veterinary science department at North Dakota State University for inoculation into immune and nonimmune sheep. Following inoculation, the unvaccinated sheep showed a scab-like lesion at the site of inoculation, while the vaccinated animals showed only slight inflammation. These findings were felt to be indicative of contagious ecthyma.

Part of the retained material was cultured in primary rhesus monkey kidney cells in an attempt to isolate the filterable virus reported as the causitive agent of contagious ecthyma.[1] The diluted material was treated with penicillin, streptomycin, and nystatin (Mycostatin®) in concentrations satisfactory for tissue culture. This was inoculated into monkey kidney cells at the rate of 0.1 ml. per tissue culture tube until all that remained had been used. These tubes were allowed to incubate in a horizontal position at 36° C. for seven days. The cells were observed for possible cytopathogenic effects at daily intervals for seven days. Then all cells were pooled and held for second passage. Cytopathogenic effects in first passage were minimal and consisted only of a slight granular appearance with some moderate rounding of the cells. Second passage was started in a different lot of the same cell line. Cytopathogenic effect was much more pronounced, the cells showing a good deal of granulation by the third day, with rounding. By the fourth day, cells were beginning to slough off, and those remaining showed almost complete degeneration. These cells were pooled, frozen, and stored at –20° C. for later identification. Some months later, a part of the pool was sent to the Communicable Disease Center in Atlanta, Ga., for identification. To date, no report has been received.

Nonimmune lambs were inoculated with pooled tissue cultures in an attempt to transmit the virus. Unfortunately, we were unable to demonstrate any clinical evidence of disease in the lambs tested. There is, however, a possibility that these lambs could have had previous infection with contagious ecthyma.

In 1961, Nagington and Whittle[1] reported cytopathogenic effects in human amnion and secondary monkey kidney cells with a characteristic cytopathogenic effect. It was their experience that subsequent inoculation of infected tissue culture cells into nonimmune sheep did not always result in clinical lesions of contagious ecthyma and that tissue culture passage tended to lower virulence.

COMMENT

In 1923, the etiology of ecthyma contagiosum, also known as orf disease, was shown to be a filterable virus.[2] The disease is fairly common among sheepherders, veterinarians, and employees working in agricultural experiment stations. Butchers also have developed lesions.[3,4] Infections have been reported from Europe, New Zealand, and Australia, as well as the United States.[3–10] Despite its wide geographic distribution, relatively few infections of man have been reported in the medical literature, but such occurrence is felt to be more frequent than the paucity of reported cases would suggest.

Infections in man occur as a result of inoculation of infected material into minor scratches and abrasions of the skin. Lesions most commonly are seen on the hands, fingers, wrists, and face.[3] Lesions on the legs have been noted.[11] The incubation period varies from a few days to a week. Skin lesions last from three to four weeks and undergo a definite pattern of evolution. Initially, macular lesions appear, progressing to papules, vesicles, pustules, and, finally, scabs which drop off, leaving no scars. Lesions may be painful, and there may be swelling of surrounding soft tissue. Regional lymphadenitis may be present, and it was noted in both of our cases. No other constitutional symptoms have been reported unless there is secondary infection of the lesions.

Direct contact with infected animals is the usual mode of transmission of the disease to man. A skin lesion appearing after an accidental scratch on a contaminated barbed wire fence has been reported.[10] Although scabs from lesions of sheep containing the virus may contaminate pastures and remain viable from one season to the next, causing new outbreaks in sheep, no lesions of man from contaminated soil have been reported. Infection of man from contaminated soil would seem a distinct possibility, particularly in light of the report of a lesion resulting from contact with a contaminated fomite, as previously noted.

Animal infection with ecthyma contagiosum is limited to sheep and goats. Lesions occur on lips, nostrils, and eyes of infected animals. Infection commonly occurs in young kids or lambs 4 to 6 months of age. Older animals develop immunity from previous infection or vaccination. Painful lip lesions prevent the infected sheep from eating, resulting in rapid weight loss. Death may occur from starvation. In these cases, there is usually secondary invasion of lesions with the screw worm fly (Cochliomyia americana) of infection with necrosis bacillus (Actinomyces necrophorus).[12–14] Sheep infections resolve in approximately one month when the last scabs fall off, leaving no scars.

SUMMARY

Two cases of contagious ecthyma in farm workers were observed. Both patients developed typical skin lesions of the hands after working with a flock of sheep known to have sore mouth disease.

Fluid from these lesions was essentially free from pathogenic bacteria and fungi. Disease in lambs, clinically typical of contagious ecthyma, was produced by inoculating vesicular fluid from one of the patients. A cytopathogenic agent was isolated from the vesicular fluid. It was impossible to demonstrate contagious ecthyma by inoculation of second passage tissue culture into lambs.

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Cerebral Abscess and Cyanotic Congenital Heart Disease

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THE DEVELOPMENT of a cerebral disorder in any child over 2 years of age with cyanotic congenital heart disease suggests a brain abscess until proved otherwise. This clinical doctrine is widely recognized by those experienced in cardiac disorders in childhood, but perhaps less so among physicians generally. The pitfalls of diagnosis in such children are obvious. Handy explanations for neurologic abnormalities may include bacterial endocarditis with embolization or cerebral vascular thrombosis secondary to polycythemia and increased blood viscosity. The occasional tendency for the manifestations of brain abscess to appear abruptly also may deceive one and again suggests a vascular occlusive problem because of the mode of onset. Even the underlying cardiac disease may so distort the important physical signs to further complicate appropriate analysis of the problem. Funduscopic examination, for example, may be more difficult to assess, because venous congestion and tortuosity are often found with cyanotic heart disease without intracerebral complications. General signs of infection, such as fever or leukocytosis, may be absent, and specific localizing signs on neurologic examination are not always present. Nevertheless, there are accurate means of establishing a clinical diagnosis of cerebral abscess if the possibility is entertained. Brain abscess occurring as a complication of cyanotic congenital heart disease is usually a solitary lesion occurring in the cerebrum and thus is potentially curable if early diagnosis is made.[1-4] If a brain abscess goes unrecognized, death is apt to occur from tentorial herniation with brainstem compression or following rupture into the ventricle with subsequent meningitis.

The following two cases are representative of the clinical features and diagnostic aspects of this disorder. In both, brain abscess developed in the absence of bacterial endocarditis or obvious suppurative processes elsewhere.

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CASE REPORTS

Case 1. R. F., a 9-year-old, right-handed male, was admitted to the University Hospitals on May 27, 1966, for evaluation of right focal motor seizures and right hemiparesis. He had previously been seen at this hospital in July 1957 at 4 months of age. Approximately two months before, he had developed wheezing respiration, but without cyanosis. Findings on the initial visit in 1957 included a loud systolic murmur and palpable thrill located in the fourth intercostal space along the left sternal border. Chest X-ray showed evidence of left ventricular hypertrophy. The pulmonary outflow tract appeared prominent, and increased pulmonary vascular markings were present. An intracardiac, left-to-right shunt was suspected, but a specific diagnosis was not established.

The child returned in April 1958, at age 1, for cardiac catheterization which revealed a patent ductus arteriosus. On May 2, 1958, thoracotomy was done and the ductus ligated. One of several dilated bronchial arteries arising just distal to the patent ductus was also ligated. On discharge from the hospital, he was described as doing well but was slightly cyanotic.

Thereafter, he was seen at six-month intervals and was noted to have increasing cyanosis, clubbing, and right ventricular enlargement. Cardiac catheterization and angiocardiography were repeated in April 1960. The pulmonary artery was not visualized; however, filling of the aorta from the right ventricle was described. The impression thereafter was either tetralogy of Fallot or truncus arteriosus.

The child did well until May 27, 1966. On this date, he arose during the night but was unable to return to bed alone because of motor dysfunction of the right leg. Within a few minutes, he experienced involuntary jerking movements of the right arm and leg lasting about three minutes. Two similar episodes occurred in brief succession and were followed by weakness of the right arm and leg. During this period, an expressive speech deficit also was described. The right-sided paresis persisted for approximately three hours. During the subsequent days, he complained of occasional headaches and experienced several episodes of vomiting.

On admission to the hospital on May 27, 1966, he was initially afebrile but within four hours had a temp-

erature of 102.6° F. His pulse was 68 per minute and blood pressure was 126/76 mm. Hg. He appeared alert, comfortable, and walked without difficulty. A loud systolic murmur was heard diffusely over the precordium. The initial neurologic examination revealed no weakness, sensory deficits, or pathologic reflexes. The deep tendon reflexes were normal. Funduscopic examination revealed the disks and adjacent vessels to be normal. Laboratory findings included a normal urinalysis. Hemoglobin was 17.3 gm. percent, hematocrit 53 percent, and white blood cell count 7,400. The erythrocyte sedimentation rate was 8 mm. per hour. Several blood cultures resulted in no growth. Biventricular cardiac enlargement with normal lung fields was seen on chest X-ray. Skull X-rays were not remarkable except for evidence of ethmoid and maxillary sinusitis. Lumbar puncture on the day of admission showed clear fluid with an opening pressure of 175 mm. of water in the horizontal position. The cerebrospinal fluid revealed no cells; sugar was 60 mg. percent and protein was 50 mg. percent.

On the fourth hospital day, the child was noted to have mild weakness of the right upper extremity and increased reflex activity of the right leg. The plantar response on the right was extensor. Electroencephalography was abnormal, with high-voltage delta activity over a large part of the left cerebral hemisphere but oriented somewhat posteriorly. Brain scan outlined an area of increased uptake of radioactive mercury in the left parietal region near the midline (Fig. 1). Before the studies he had been placed on intravenous penicillin, chloramphenicol, and intramuscular streptomycin.

On June 2, 1966, he complained of vomiting and severe headaches. Throughout the day he remained lethargic. On June 3, generalized twitching movements developed, followed by intermittent episodes of rigid extension of the limbs and hyperextension of the trunk and neck. Lumbar puncture was repeated, and the opening pressure was 450 mm. of water in the horizontal position. The fluid was turbid; segmented neutrophils filled the counting chamber. The CSF protein was 420 mg. percent and sugar was zero. Left carotid angiography was done soon thereafter on the same day (Figs. 2-4). The left anterior cerebral artery and the internal cerebral veins were shifted across the midline to the right. An avascular mass, deep and near the midline, was present in the left parietal lobe. Rapid deterioration occurred, and the child died on June 4, 1966, on the ninth hospital day.

Postmortem examination verified the clinically suspected diagnosis of an intracerebral abscess. A single abscess was found in the left parietal lobe just beneath the cortex in the precuneus. The lesion measured approximately 3 cm. in diameter and had ruptured into the left lateral ventricle producing purulent meningitis. Cultures from the abscess and of the cerebrospinal fluid remained negative. There was no evidence of endocarditis or pulmonary infection. Multiple intrathoracic

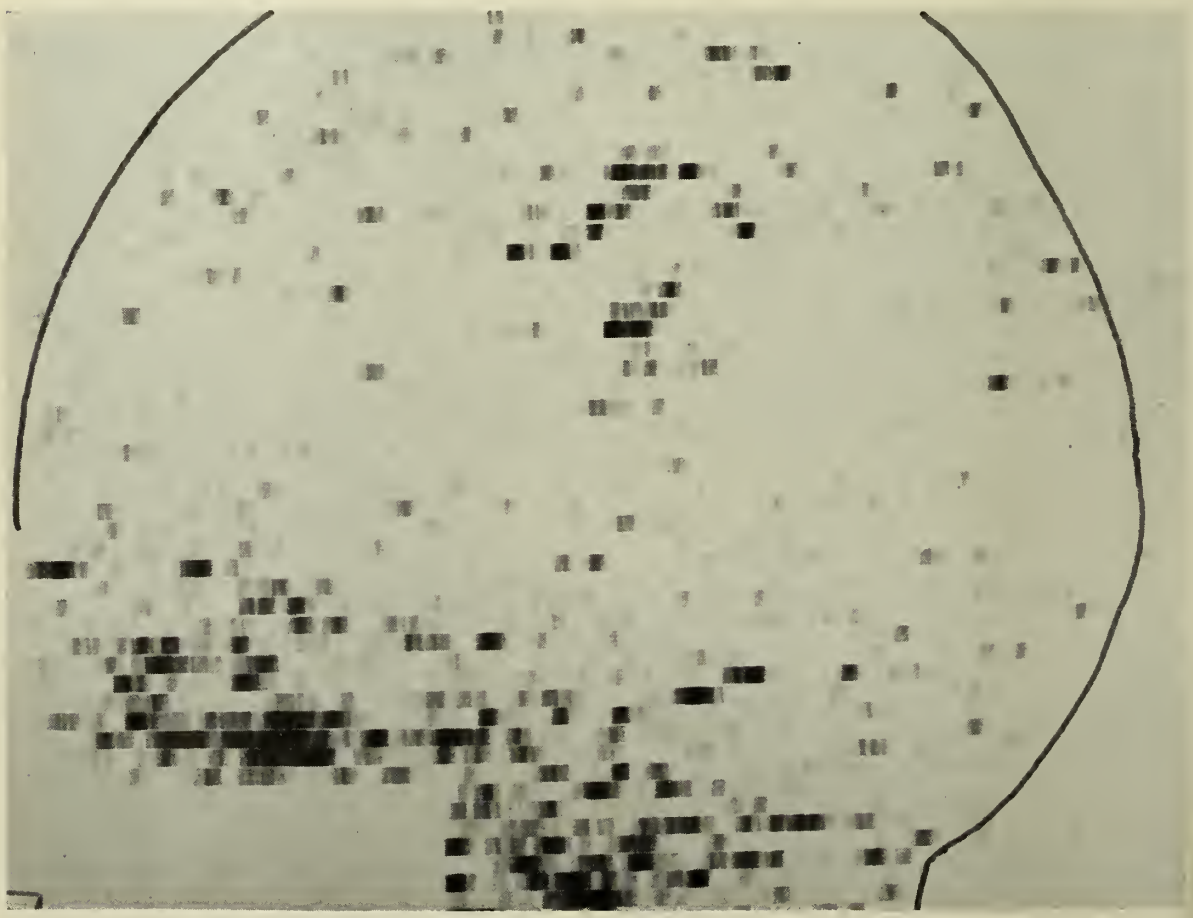


Fig. 1. (Case 1.) Radioactive brain scan showing abnormal uptake in the region of the abscess



Fig. 2. (Case 1.) Left carotid angiogram, lateral view. Vessel indicated by arrows on stereoscopic view is displaced laterally, suggesting underlying abscess.



Fig. 3. (Case 1.) Left carotid angiogram, anteroposterior view, showing displacement of the anterior cerebral artery across the midline

anomalies were found, including atresia of the right and left pulmonary arteries. Cardiac enlargement was present with a large ventricular septal defect.

Case 2. M. S., a 9-year-old girl, was admitted to the University Hospitals on Jan. 30, 1964, following the onset of right focal motor seizures three days before. She had been seen at this hospital in December 1959 at age 5 for evaluation of a heart murmur. During early childhood, frequent upper respiratory infections and ease of fatigue had occurred. Examination in December 1959 revealed a moderately loud systolic murmur and a palpable thrill present in the left third intercostal space. The liver was felt 4 cm. below the right costal margin. The lips were slightly dusky, and mild cyanosis of the nailbeds was noted. Chest X-ray showed generalized cardiomegaly with findings consistent with pulmonary hypertension. An electrocardiogram showed right ventricular hypertrophy and P-wave abnormalities suggestive of atrial disease. Cardiac catheterization was performed, and it was believed to show evidence of pulmonary hypertension and an intracardiac right-to-left shunt. The child improved after digitalization. On periodic out-clinic examinations, she continued to have moderate cyanosis and, except for decreased exercise tolerance, did reasonably well.

On Jan. 27, 1964, while at school, she noticed weakness of the right arm. Two hours later, she developed twitching movements of the right hand with progression up the right arm to the shoulder. She experienced several such episodes, each lasting about two minutes and without loss of awareness. The following day, the child had nausea, vomiting, and headache and again had a right focal seizure. This episode began in the right foot and then extended up the leg to involve the right arm and the face on the right. She was admitted to the local hospital where she was found to be drowsy and to have weakness of the right arm and leg. The following day, the patient was transferred to the University Hospitals.

On admission, she was alert but appeared dehydrated. Moderate cyanosis was present. Temperature was 102.4° F., pulse 132 per minute, and blood pressure 80/60 mm. Hg. Examination of the heart showed overactivity and a loud holosystolic murmur in the third left intercostal space along the sternal border. Clubbing of the fingers was marked. Pupils reacted normally to light, and funduscopic examination was normal. A right central facial paresis was present in addition to moderate weakness of the right arm and leg. Sensory examination was normal. The plantar response on the right was extensor.



Fig. 4. (Case 1.) Left carotid angiogram, venous phase, showing displacement of the left internal cerebral vein across the midline

Laboratory findings on admission included a normal urinalysis. Hemoglobin was 15.8 gm. percent; hematocrit, 48 percent; and white blood cell count 11,750 with 83 percent neutrophils, 13 percent lymphocytes, and 4 percent monocytes. Serum electrolytes and urea nitrogen were normal. Several blood cultures yielded no bacterial growth. Chest X-ray showed right ventricle enlargement and prominent pulmonary outflow tract. Lumbar puncture was performed with the opening pressure being 255 mm. of water in the horizontal position. The fluid was clear, 4 lymphocytes per cubic millimeter were present. The CSF sugar was zero and protein was 62 mg. percent. CSF culture was negative.

Headaches persisted after admission; however, the child became afebrile, and the right hemiparesis rapidly improved. An electroencephalogram was abnormal with focal activity over the left central parietal region. Thereafter, she was placed on intravenous penicillin and sulfadiazine therapy. On the fourth hospital day, headache became more intense, and weakness of the right side recurred, lethargy became more pronounced, and the child continued to complain of headache and nausea. The pulse rate was found to be 56 per minute, and venous engorgement was apparent on funduscopic examination.

On Feb. 10, 1966, left carotid angiography was performed and showed an avascular mass estimated to be 6 cm. in diameter in the left posterosuperior parietal region (Figs. 5 and 6). The child was taken to the operating room, and a left parietal craniotomy was performed. An intracerebral abscess was identified 1.5 cm. beneath the cortex in the parietal region, and 40 cc. of purulent material were drained through a ventricular needle. The abscess was then dissected free and removed. Smear of the material aspirated from the abscess showed gram-positive streptococci. Intravenous antibiotics were continued postoperatively. Two weeks after surgery, she suddenly became unresponsive and soon thereafter died.

Postmortem examination revealed no obvious explanation for the sudden death. The atrium and right ventricle were markedly dilated. A high atrial septal defect was present in addition to enlargement of the pulmonary artery. No evidence of bacterial endocarditis was found. The left cerebral hemisphere appeared large on gross observation. An abscess cavity was present in the left parietal region just below the cortex and extended just anterior to the region of the splenium of the corpus callosum.

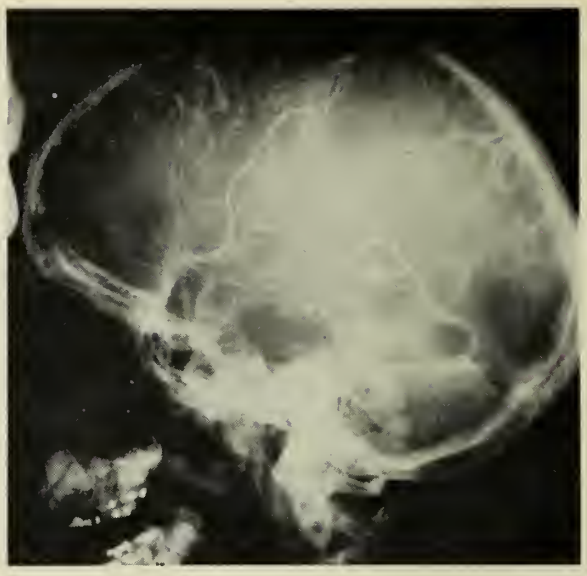


Fig. 5. (Case 2.) Left carotid angiogram, lateral view, venous phase. An abnormal accumulation of dye, presumably in the abscess capsule, is noted in the parietal region.



Fig. 6. (Case 2.) Left carotid angiogram, anteroposterior view. A marked shift of the anterior cerebral artery across the midline is seen.

DIAGNOSTIC ASPECTS

In both patients described above, the neurologic manifestations resulting from the intracerebral abscess had an abrupt onset. However, both had focal motor seizures and both experienced headaches soon thereafter. Regardless of the presence or absence of other findings, these clinical features in patients with heart disease must be regarded as suggestive evidence of brain abscess. The hazard of delay in diagnosis and surgical management is evident in Case 1. Here, rather rapid progression of the disorder occurred, and spontaneous rupture of the abscess into the lateral ventricle resulted in fulminating bacterial meningitis and death.

Brain abscess may occur in children with a variety of conditions; however, infectious states such as mastoiditis or pulmonary empyema have become less common causes. Cyanotic congenital heart disease remains as the single common correlate with brain abscess formation in the childhood years. This cerebral complication may occur with several types of intracardiac anomalies. It is especially prevalent with those with right-to-left shunts resulting in significant

amounts of poorly oxygenated venous blood recirculating through the systemic circulation, thus bypassing the filtering effect of the lungs.[3,5] Further support for the importance of the filtering effect of the pulmonary bed is evidenced by cases of cerebral abscess associated with pulmonary arteriovenous fistula.[6,7] Tetralogy of Fallot with a major right-to-left shunt is the most common congenital cardiac lesion in which the patient has a reasonable chance of survival for more than two years. As expected, this is the anomaly most commonly associated with brain abscess formation.[2,8–10] Surgical procedures, such as the Pott's or Blalock anastomoses which improve hemodynamics, do not necessarily exclude the possibility of subsequent development of a brain abscess. These anastomotic procedures increase pulmonary blood flow, but the right-to-left shunt at the ventricular level persists, permitting venous dilution of systemic blood. Clark and Clarke[2] described seven patients with brain abscess developing after surgery for tetralogy of Fallot. Wishingrad and associates[11] recently reported a cerebral abscess occurring in a child 7 years after a Pott's procedure for tetralogy of Fallot. While severe grades of desaturation may occur with transposition of the great vessels, the limited life-span provides far fewer patients likely to develop brain abscess. The great majority of cases of brain abscess with heart disease occur in children over 2 years of age.[12] The reason for this remains unclear, but perhaps the infrequency of bacteremic episodes in infancy compared with the older child provides a partial explanation. The infant group is not totally immune, however. Raimondi and co-workers[13] described a baby who died at 20 days of age with congenital heart disease and a parietal lobe abscess.

In addition to a right-to-left shunt, other factors are important in the pathogenesis of brain abscess. A source of bacteria is necessary; however, this is not usually related to bacterial endocarditis. Often the preceding infection is barely perceptible or unidentified.[12,14–17] At times, definite evidence of carious teeth, carbuncles, stomatitis, or pharyngitis[18] is present. Other sources of infection described have included dental extraction,[19] pharyngeal abscess,[20] and osteomyelitis with liver abscess.[5] There is some evidence to support the concept that brain abscess is prone to occur in an area of brain tissue previously damaged by ischemia or vascular occlusion.[15,21,22] Groff[23] showed that in animals bacteremia did not result in abscess formation in the brain unless preceding brain tissue injury had occurred. Persistent hypoxia, polycythemia, and increased blood viscosity in children with cyanotic heart disease predispose to such ischemic cerebral insults and provide fertile soil for subsequent abscess formation.[4,5,8,10,14,18,24,25]

Available data on the frequency of brain abscess with heart disease is mainly from study of autopsy material. Hanna[8] found on examination of 160 autopsied cases of congenital heart disease that brain abscess was present in 6 (3.7%). Maronde[12] identified 11 (5%) in 209 autopsied cases of congenital heart disease. Fontana and Edwards[26] did pathologic studies on 357 patients with major cardiac malformations and found 10 (2.8%) with brain abscess; none showed evidence of bacterial endocarditis. Other attempts at estimation of the frequency of this cerebral complication with congenital heart disease included that by Robbin,[15] who found 3 brain abscesses in 53 patients (5.6%) with congenital heart disease.

The symptoms and signs in children with brain abscess show considerable variation and in part are determined by the side and site of the brain involved. Blood-borne abscesses are often found in the distribution of the middle cerebral artery. Otogenous abscesses are prone to develop in either the temporal lobe or the cerebellum. Thus, the cerebral lesions in children with cyanotic congenital heart disease are usually parietal or parieto-temporal in location. The onset of symptoms is often gradual, but progression of abnormalities is common in the subsequent days. In the patients described above, the first manifestations were abrupt, but the subsequent progressive features served as points of differentiation from embolic or thrombotic lesions. Headache and vomiting are usual and in one series[13] were described in 15 of 19 children with cerebral abscess. Seizures may be either generalized or focal and often recur at frequent intervals. Bruno[27] reported seizures in 36% of patients with brain abscesses. Newton[3] found convulsive manifestations in 35% and focal seizures in 19% of patients with brain abscess with cyanotic congenital heart disease. Recurrent focal convulsions in an older child with congenital heart disease must be considered uncommon with vascular insults and should suggest abscess. Hemiparesis, hemianoptic field defects, dysphasia, and lethargy are additional common manifestations.[28] Brain abscesses commonly provoke considerable edema of surrounding brain tissue. Increased intracranial pressure may be reflected by papilledema and diplopia, but their absence need not exclude the possibility of brain abscess. Evidence of active infection may not be present.[10] In the series reported by Raimondi and associates,[13] temperature over 101° F. was noted in only 6 of 19 pa-

tients with brain abscess. Blood leukocytosis may be absent unless meningitis coexists. Schurr[25] has attempted to correlate clinical findings with three phases of cerebral abscess formation. The initial stage is one of bacterial encephalitis with malaise, headache, and slight fever but without localizing signs. In the second phase, the inflammatory process becomes circumscribed and some general improvement of symptoms may occur. The third stage consists of enlargement of the abscess and capsule formation. At this time, manifestations of increased pressure plus focal neurologic abnormalities appear. He suggests that four to six weeks is the minimum period needed for the formation of a firm capsule surrounding the abscess.

Skull X-rays are frequently normal in children with brain abscess unless the child is relatively young and suture spread occurs as a result of increased pressure. Generalized diploic thickening of the bones of the skull has been described in children with cyanotic congenital heart disease.[29] This most likely results from chronic hypoxia with compensatory bone marrow hyperplasia and should not be confused with an abnormality related to increased intracranial pressure. Lumbar puncture and CSF examination may reveal no abnormalities.[14,28,30,31] As a rule, however, the opening pressure is elevated, the fluid is clear and colorless, and the culture is negative. A lymphocytic pleocytosis is often present. Five of thirteen cases described by Matson and Salam[10] had significant cellular responses ranging from 88 to 290 cells per cubic millimeter. Should leakage or rupture of the abscess into the subarachnoid space or ventricle occur, the cellular response would be greater and the CSF sugar would be decreased. Laboratory error must also always be considered as a potential explanation for inconsistent results and probably accounts for the CSF sugar of zero in Case 2 described above. Electroencephalography is of highly localizing value for brain abscess[10,32,33] if the lesion is located in one cerebral hemisphere. Cerebellar lesions may produce no electrical abnormalities or those secondary to generalized increased pressure. As opposed to ischemic lesions where more generalized slowing is common, cerebral abscesses are often associated with localized high-voltage delta activity. In one series of 41 brain abscesses with preoperative EEGs,[34] only two recordings were described as normal. The EEG was considered "specific" in 29 of 41. Accurate localization of the lesion occurred in 17 and lateralization in 12. Radioactive brain scan technique has recently been added as a potential diagnostic aid in brain abscess.[35–38] At present, sufficient data has not accumulated to warrant a decision as to its value in this condition. Carotid angiography would appear to be the preferred contrast study for localization in most instances of cerebral hemisphere abscesses.[13] Concern for the hazard of angiography[10] in the presence of increased blood viscosity has been raised. However, the relative lack of change of intracranial pressure dynamics with angiography as compared with ventriculography would favor the former as the usual procedure of choice. Clinical evidence suggestive of cerebellar involvement would be more profitably investigated with either ventriculography or cautious fractional pneumoencephalography.

DIFFERENTIAL DIAGNOSIS

Cerebral vascular thrombosis is a recognized common complication of cyanotic congenital heart disease and may be difficult to distinguish from brain abscess. The occlusive episode may appear to occur spontaneously or may be precipitated by diarrhea or a febrile disorder with associated mild dehydration.[39] Berthrong and Sabiston[5] identified cerebral infarctions due to vascular occlusion in 18.5% of 135 autopsied cases of cyanotic heart disease. Martelle and Linde[9] noted an incidence of cerebrosvascular occlusions in 10% of 50 patients with tetralogy of Fallot. Banker[40] reported a 10.2% incidence of cerebral occlusive disease in 108 children with congenital heart disease. Cohen and Bergman[24] studied 100 fatal cases of congenital heart disease and suggested that chronic circulatory inadequacy without specific thrombotic lesions accounted for many of the ischemic cerebral changes. Others[41] have also postulated cerebral infarction in the absence of vascular thrombosis on the basis of pathologic studies. Cyanotic children with cerebral thrombosis are prone to have a more precipitous onset than are those with abscess. As noted above, however, either may be initiated by a convulsion. Thrombotic lesions are more characteristic of children under two years, while cerebral abscesses occur more often in older children with heart disease. The clinical course and ancillary diagnostic procedures usually distinguish the two conditions. Occasionally, angiography may be necessary when one cannot otherwise exclude brain abscess.

Bacterial endocarditis with its attendant cerebral complications may also cause diagnostic confusion. Characteristic findings of endocarditis, including anemia, fever, increased sedimentation rate, splenomegaly, and cutaneous petechiae, simplify the diagnosis. However, such clear-cut evidence is not always apparent, and neurologic

abnormalities may be the initial manifestation of endocarditis. Cerebral complications of bacterial endocarditis include vascular occlusion with infarction, mycotic aneurysms, or meningitis. Rupture of a cerebral mycotic aneurysm may result in sudden onset of findings indicative of intracerebral or subarachnoid hemorrhage. Angiography is necessary for identification and localization of such lesions and to exclude brain abscess.

Complications of endocardial fibroelastosis might also be confused with brain abscess. While not usually classified as a type of cyanotic congenital heart disease, endocardial fibroelastosis often has its clinical onset in early infancy. Branch and Castle[42] reported five children with this type of endomyocardial disease with evidence of cerebral embolization, probably arising from mural thrombi in the heart. It is of some interest that four of the five children had a significant hypochromic, microcytic anemia. A similar finding has been noted[9] in patients with cerebral occlusive lesions with tetralogy of Fallot. As with other embolic states, sudden onset, usual lack of progression, and absence of increased intracranial pressure distinguish it from brain abscess.

Evidence of a mass lesion with symptoms of increased intracranial pressure and focal signs may suggest the possibility of a cerebral neoplasm. The more rapidly progressive clinical course of a brain abscess, plus the difference of the spinal fluid abnormalities, aids in differentiation of the two lesions. Patients with brain abscess who have fever and a lymphocytic cellular response in the spinal fluid may be considered to have viral encephalitis initially. The progressive course, the appearance of papilledema, and the development of localizing neurologic signs become more consistent with a focal suppurative cerebral process than with a more generalized infectious disorder. Tuberculous meningitis may result in a more difficult differential diagnostic problem due to the similarity of clinical features of this disorder with cerebral abscess. Evidence of more diffuse involvement is usually present with tuberculous meningitis, including multiple cranial nerve palsies.

TREATMENT

Preoperative management of children with brain abscess includes efforts to control or prevent seizures and antibiotics to confine the infectious process. Equally important are considerations to avoid potentially harmful measures which might make surgical therapy less beneficial. For example, if increased intracranial pressure is present, one should avoid narcotics and refrain from the use of enemas. Intravenous fluid therapy in excess of what is necessary may contribute to further cerebral edema and lead to tentorial herniation. Unless the child is significantly dehydrated, maintenance fluid therapy may be calculated on the basis of 1,500 cc. per square meter per twenty-four hours. Electrolyte-free solutions should be avoided, as any tendency to hyponatremia may also aggravate cerebral edema. Intravenous fluids are usually necessary preoperatively in children with cerebral abscesses because of the frequency of vomiting and to provide a route for antibiotics in large dosage. As staphylococci or streptococci are the most common organisms identified from cerebral abscesses, penicillin would appear to be a reasonable initial drug of choice. If penicillinase-producing staphylococci are identified from aspirated material at surgery, a more appropriate preparation such as methicillin should be substituted.

Neurosurgical treatment of brain abscess in children with congenital heart disease may consist of burr hole and needle drainage or craniotomy and attempted resection of the lesion as a whole. As the degree of capsule formation can rarely be accurately estimated preoperatively, perhaps initial drainage of the abscess through an appropriately placed burr hole provides the safest approach in the hands of most. This permits readjustment of disturbed intracranial dynamics, an estimation of capsule thickness, and, if necessary, provides additional time for capsule development before more definitive resection. At the same time, it avoids the hazard of spontaneous rupture of the abscess which may occur by temporizing until one estimates that the capsule around the abscess has formed adequately for total resection as the primary procedure. In certain instances, however, primary total removal of the abscess and its capsule would be in order. If the lesion is relatively small, if one can be certain that cerebritis surrounding the lesion is minimal, and if the general condition is adequate, primary resection may be advisable.

SUMMARY

Children with cyanotic congenital heart disease are subject to the development of cerebral abscesses because of the peculiar anatomic and physiologic features of the cardiac disease. Chronic hypoxia may produce cerebral lesions that serve as a site for growth of organisms. Compensatory polycythemia with increased blood viscosity tends to slow cerebral circulation. The intracardiac shunt permits systemic ac-

cess of venous blood bypassing the filtering effect of the lungs. Transient bacteremia thus provides a greater hazard for such children and may lead to intracerebral suppuration. The onset of symptoms is usually gradual but may appear abruptly with convulsions as described above. Consideration of the possibility of brain abscess with appropriate diagnostic studies usually allows for accurate diagnosis of the disease, proper localization of the lesion, and institution of appropriate therapy.

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Allopurinol in the Treatment of Gout

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The therapy of gout, as with many other diseases, has changed during the past decade. The single method restricted to treating the recurring acute attack has given way to the use of prophylactic treatment to prevent permanent disability either from erosion of bone and joint cartilage or from renal damage. The long-term management of gout can usually be achieved by giving uricosuric agents to lower the serum urate and to prevent and treat tophaceous gout. The 2 most widely used uricosuric agents of recent years are probenecid (Benemid®) and sulfinpyrazone (Anturane®) which act to lower the serum urate level by suppressing tubular reabsorption of the urates and thereby increase urinary urate excretion. Although these drugs are satisfactory in the majority of gouty patients, there are always some people in which these uricosuric agents cannot be used because of drug intolerance or impaired renal function. With moderate to severe impairment of renal function, uricosuric agents of all types are ineffective and even contraindicated because of the additional hazard of urolithiasis and even obstruction of lower urinary tract. This is especially true in patients with neoplastic disease of the hemopoietic system, with or without secondary gout, whose nucleic acid turnover is so increased and serum urate so high that a great amount of uric acid is fed into the urinary tract on treatment of the proliferative disorder. Therefore, uricosuric agents should be avoided in patients with diminished renal function, in those who have a tendency to urolithiasis, and in those who have severe secondary gout. Fortunately, recent studies have demonstrated a drug that can be used in these difficult situations.

From studies to attempt to increase the therapeutic value of 6-mercaptopurine (6-MP) and similar thiopurines in leukemia, allopurinol (Zyloprim®)—4-hydroxypyrazolo (3, 4-d) pyrimidine (4-HPP)—was discovered as a new and useful drug in the treatment of gout. 6-MP in the mouse[1] is oxidized and converted to the inert metabolite, 6-thiouric acid, which is excreted in the urine. The enzyme responsible for this oxidation is xanthine oxidase, the same enzyme that converts the normal purines, hypoxanthine and xanthine, to uric acid. Therefore, a useful tool in 6-MP therapy would seem to be a xanthine oxidase inhibitor. Elion and associates[2] chose 4-HPP and found that it not only greatly reduced the therapeutic dose of 6-MP in patients with leukemia by inhibiting 6-MP oxidation but that it also inhibited uric acid production. This 4-HPP or Zyloprim®, an inhibitor of xanthine-oxidase which prevents conversion of hypoxanthine to xanthine and of xanthine to uric acid, was found to be a new approach in the control of gout.[3–5] The uric acid formation is decreased and the uric acid available for excretion is thereby diminished, while the more soluble xanthines are readily excreted, which permits the use of Zyloprim® in the presence of impaired renal function.

Therefore, since it is now possible to both decrease the formation of uric acid (Zyloprim®) and to augment its urinary excretion (uricosuric, usually Anturane®), it is logical to use these drugs combined.[6,7] With combined therapy, smaller daily doses of each drug can be used which decrease the incidence of toxic side effects from the larger doses of the individually used drug. Also in tophaceous gout, if only Zyloprim® is used the rate of disappearance of the urate deposits will be slower than if a concurrent uricosuric agent is used to pull uric acid out of the tophi into the tissue fluids.[4,6–10] Of course, although Zyloprim® can be used alone with decreased renal function, combined therapy can only be used in the presence of adequate renal function.

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MATERIAL AND METHODS

Forty-two patients were selected for clinical study, 22 treated with allopurinol alone and 20 under combined therapy with Zyloprim® and Anturane®. They have been under treatment in the clinic or private office from one month to eight years and have often been seen by more than 1 physician.

Serum uric acid and urine uric acid determinations were done by the enzymatic method.[11] Urinary xanthine determinations were done by a modification of the method of Kalckar.[12] The oxypurines were reported by calculation as milligrams of xanthine, with no attempt to identify the actual percentage derived from hypoxanthine.

Many of the patients were subjected to extensive laboratory testing before and during the clinical trial. The tests included complete blood count, erythrocyte sedimentation rate, urinalysis, serum uric acid, urinary uric acid, urinary oxypurines, serum glutamic oxalopyruvic transaminase, cephalin cholesterol flocculation, thymol turbidity, alkaline phosphatase, creatinine, bilirubin, cholesterol, alpha- and beta-lipoprotein-cholesterol, serum protein electrophoresis, and blood urea nitrogen.

Observations were made at variable intervals depending upon the seriousness of the clinical problem considered. Measurements were made of visible tophi metrically, photographically, and radiographically.

RESULTS OF ZYLOPRIM® ALONE

Twenty-two patients (11 female) were treated from 42 to 486 days or a total of 5,680 patient-days with Zyloprim® alone.

A. *Impaired renal function and urolithiasis.* Two of the subjects in this group had previously been unable to take uricosuric agents because of severely impaired renal function. Each had multiple tophi which had been steadily increasing in size. One patient, aged 85, was able to tolerate from 400 to 600 mg. daily for 270 days during which his serum uric acid diminished from 11.35 to 5.40 mg. per 100 ml. His serum creatine was 2.5 to 4.5 mg. per 100 ml. before and during treatment. The tophi became smaller and softer and his range of general physical activity increased. The second patient has continued to take from 300 to 500 mg. daily for 486 days while her serum uric acid has diminished from 8.98 to 5.74 mg. per 100 ml. Her tophi have not increased in number, and some of them have become softer and smaller. She has experienced less pain and the renal function has remained unchanged.

Two other patients had previously passed many urate stones. In both, Anturane® previously had prevented attacks of acute gout and lowered the serum uric acid, but stones continued to form. During this therapeutic trial on Zyloprim® neither patient passed stones.

B. *Secondary gout.* A male patient with chronic myelogenous leukemia could not tolerate Anturane® because of "flushing sensation" and nausea. On Zyloprim® of 50 to 300 mg. without nausea he was controlled but had a massive gastrointestinal hemorrhage from a duodenal ulcer. After treatment of the ulcer and leukemia with P_{32} with good effect, he was able to resume the medication.

Another male patient with chronic lymphatic leukemia with a large spleen and liver was able to decrease his uric acid to 6.5 to 7.5 mg. per 100 ml. with sulfinpyrazone but passed many small urate stones. On 200 mg. allopurinol his uric acid has been normal for seven months and no stones passed.

C. *Toxic reactions to sulfinpyrazone.* Three patients previously unable to tolerate sulfinpyrazone because of vertigo, rash, nausea, and transient leukopenia were treated with Zyloprim® from 42 to 181 days. Elevated serum uric acid became normal in all of them during treatment. In 2, medication was well tolerated, but the third was forced to discontinue medication after 42 days because of nausea, diarrhea, and furunculosis of the nose.

D. *Psoriasis and psoriatic arthropathy.* Two patients with psoriatic arthropathy were treated with Zyloprim® 200 mg. daily because of hyperuricemia and experienced reduction of their uric acid to normal under treatment. One had marked diminution of pain and swelling of the involved joints and the other, after treatment for 72 days, also showed reduction in the skin area involved by psoriasis.

E. *Miscellaneous.* Five patients with duodenal ulcer were treated with Zyloprim® with the ulcers remaining quiescent in all and the uric acid becoming normal. Previously, 4 of these had been unable to tolerate sufinpyrazone because of ulcer aggravation and in the fifth, uricosuric therapy was not tried because of the severity of the ulcer.

Six patients whose medical management required the use of thiazide diuretics were not controlled with Anturane®. Since receiving 100 to 300 mg. of Zyloprim® per day their serum uric acid has been lowered to normal and they have not suffered from acute gout.

COMBINED TREATMENT

Twenty male patients were treated with Zyloprim® and Anturane® in combined therapy.

A. *Without tophi.* Eight of these patients without tophi were treated for 5,323 patient-days. All had previously been on treatment with uricosuric agents alone (100 to 1,000 mg. Anturane®) and had not had normal serum uric acid. Of these, 3 had passed urate stones. On adding 100 to 400 mg. Zyloprim® to the regime the uric acid diminished to normal in 7 out of 8 patients. It was possible to cut the dose of Anturane® to 100 to 200 mg. with 100 to 400 mg. of Zyloprim® and no stones were passed. The eighth patient was only taking 100 mg. of each drug daily which probably was not sufficient medication.

B. *With tophi.* Twelve patients with tophi were treated for a total of 3,723 patient-days. These 12 patients had previously been treated with uricosuric agents and showed visible tophi which had not increased in size or which had regressed only slightly. They were then treated with Zyloprim® from 200 to 600 mg. daily. At the beginning of combined treatment, or just Anturane®, only 4 of the 12 patients showed a serum uric acid below 6.00 mg. per 100 ml. At the end of the trial 10 of the 12 had serum uric acid values below 6.00 mg. per 100 ml. and the remaining 2 were only 6.80 and 6.02 mg. per 100 ml. respectively. Eleven of the 12 showed softening and regression of tophi—only 1 showed no change in the visible tophi. Of 2 patients who previously were troubled occasionally with passing urate stones, only 1 of them passed a single stone during the treatment period.

TOXIC MANIFESTATIONS

In only 2 instances Zyloprim® had to be discontinued because of undesirable side-effects of medication. One had headaches, nausea, and vertigo after 120 days of treatment that was definitely related to taking the drug. The other, after 42 days, was forced to discontinue it because of nausea, diarrhea, and a severe furunculosis of the nose.

There was 1 incidence of leukopenia at the time of death from another cause in an 86-year-old male who was on the medication. It could not be determined whether the leukopenia was due to the drug or not.

Some had nausea or diarrhea or both, but all were able to continue the drug, some with reduced doses.

There were no laboratory abnormalities noted other than the one questionable leukopenia. Tests directed toward hepatic abnormality remained unchanged. Oxypurines were recovered from the urine in all patients on Zyloprim®.

DISCUSSION

The use of Zyloprim® as an inhibitor of xanthine oxidase activity in gout has extended the scope of treatment to include several groups of patients previously beyond the reach of therapy.

Since impairment of renal function has constituted a contraindication to the use of uricosuric agents, the availability of Zyloprim® for favorable treatment in this group, as confirmed by many,[3,5] has been a great advance in the therapeutics of gout. Although this group of patients is relatively small in relation to the total gouty population, their prior disability has often been impossible to control.

Since Zyloprim® diminishes uric acid formation, the concentration of the uric acid in the renal tubular filtrate falls to levels not readily conducive to stone formation. This observation has led to the use of the drug in groups of gouty patients who form urate stones readily, particularly when treated with uricosuric agents. Our experience in 7 patients emphasizes the importance of using Zyloprim®—only 1 of these patients passed a single stone during the period of this study.

While it is generally accepted that most gouty patients can be controlled effectively with uricosuric agents, particularly Anturane®, the addition of Zyloprim® to the treatment regimen of patients with massive tophi has resulted in hastening their softening and resolution. Combination therapy diminishes the increment in uric acid production while augmenting urinary output of uric acid. This combined therapeutic approach may be expected to "pull out" stored uric acid, making it available to excretion and thus accelerating the rate of resolution of tophi. In our 12 patients with tophi given the combined treatment this idea was substantiated by the rapid softening and shrinking of tophi in 11 instances, whereas on previous therapy with Anturane® alone the tophi were only either slowly decreasing or remaining about the same size.

There are other special problems in gout where allopurinol may be used alone in treatment. These situations include, [1] the presence of active peptic ulcer, [2] patients with toxic side reaction to uricosuric agents, [3] hyperuricemia with necessary use of thiozide diuretics, [4] psoriasis, and [5] secondary gout.[13]

The toxicity of Zyloprim® in our hands has not been alarming. Only 2 of 42 patients were

obliged to discontinue its use. Our patients have been able to achieve clinical control of gout in most instances using daily doses of 200 to 400 mg. which are less than those often employed by others. Admittedly the treatment with Zyloprim® could have been more vigorous, resulting, for example, in even more rapid resolution of tophi. Since the patients were doing well clinically this did not appear to be necessary and we feel that the use of low dosage schedules accounts for our relatively low incidence of diarrhea and of augmented attack rate of acute gout as previously reported by others.[4,5]

SUMMARY

It should be emphasized that almost all gouty patients can be safely and effectively controlled by the uricosuric agent Anturane®.

However, the new drug, Zyloprim®, when used in combination with a uricosuric agent resulted in greater diminution of serum uric acid, accelerated softening and regression of tophi, and cessation of renal stone formation.

Zyloprim® alone is the best treatment in gouty patients with impaired renal function, in patients with duodenal ulcer aggravated by uricosuric agents, in psoriasis with hyperuricemia, in patients who had toxic reactions to prior use of uricosuric agents, in secondary gout, and in patients using thiazide diuretics.

Flare-up of acute gout and toxic reactions were rare because of the low dosage schedule.

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Continued from page 396

ACUTE MYOCARDIAL INFARCTION COMPLICATED BY COMPLETE HEART BLOCK

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Patent Ductus Arteriosus

Review of Literature and Experience with Surgical Corrections

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EKSTROM,[1] in his comprehensive review of the literature, states that the ductus arteriosus was described in the second century by Claudios Galenos (Galen), in 1561 by Fallopi, and in 1564 by Arazi. Harvey, in 1628, demonstrated the importanee of the duetus in fetal circulation. Apparently, respiration is the most important stimulus to closure of the ductus under physiological conditions.[2,3] In the human being, closure probably occurs in two phases: [1] a rapid functional, reversible eontraetion of the muscular wall simultaneous with occlusion of the umbilical arteries and [2] the obliteration, by proliferation, of the conneetive tissue in the innermost layer of intima whieh can take as long as a few months for completion. Christie and Magovern[4,5] state that 65% of infants have an anatomical patency of the ductus arteriosus at the age of 2 weeks and 1%, at the age of 1 year. If functional elosure does not occur within two weeks, the ductus will probably remain patent. In 10% of the cases, patent ductus arteriosus is associated with cardiovascular anomalies.

The condition of patent duetus arteriosus was recognized clinically and correlated with autopsy findings by Gibson[6] in 1900. However, it was not until 1907 that operation was first proposed by John C. Monro of Boston.[7] Operation was suggested because [1] the patient would probably not live to puberty, [2] the lesion was relatively superficial, and [3] ligation would result instantly in permanent restoration of normal function. Monro proposed that the duetus be approached through a sternal splitting incision. Despite the logic of his proposition, Monro never succeeded in having a ease referred. According to Meade,[8] Maud Abbott had written that Monro's operation would be followed by endarteritis and, therefore, it was fortunate that no one had tried it beeause the end condition would be worse than the original one. Ashton Graybiel and assoeiates[9] interested John Strieder in the ligation of a patent ductus in a terminal patient with subacute baeterial endarteritis. This 22-year-old female was operated March 16, 1937. The ductus was firmly attached to the right pulmonary artery. Posterior freeing was thought to be very hazardous. Therefore, plieating sutures were placed, and no attempt was made to evert and suture the pulmonary artery at the pulmonary opening of the ductus. Death on the fourth postoperative day was attributed to acute dilatation of the stomach. Infarction of lungs and acute endarteritis of the pulmonary artery, not continuous with endarteritis of the ductus, were found at autopsy.

On Aug. 26, 1938, Gross and Hubbard[10] successfully ligated a patent ductus arteriosus in a 7½-year-old patient. Gross used an anterior thoracotomy approach through the third intercostal space. The thrill was obliterated but a murmur remained. The first division of a patent ductus arteriosus was attributed[1] to Craaford in 1941.

By 1944, Gross[11] recommended division rather than ligation of the patent ductus arteriosus for the following reasons: [1] Whenever a large vessel is obstructed in continuity, tiny crevices are left with the expectation of a subsequent leak. [2] Cinching down of the ligature may result in pressure erosion of the vessel wall with possible reestablishment of the vascular channel, development of a regional hematoma, or fatal hemorrhage.

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Division of the ductus arteriosus was simplified, and its safety was improved by the development of a noncutting, nonerushing clamp by Potts[12] and by subsequent modifications. Since that time numerous authorities championed the cause of division rather than ligation.[13–17] However, some still favor the ligation technique because of its greater safety, minimal recurrence rate, and belief that this procedure may parallel nature's method of obliteration.[18–24]

Bickford[25] stated that in patients whose patent ductus measured less than 7 mm. in diameter, ligation would always result in successful closure. Bauersfeld and associates[26] stated that when the ductus measured less than 10 mm., ligation was as effective as division.

Fishman and Silverthorne[27] reported one patient who had reached the age of 70 years and one who did not die with patent ductus arteriosus until she was 75 years of age. However, it should be pointed out that the elder patient died of the disease and that the younger one was a chronic invalid because of cardiac failure, probably due to the congenital lesion. One can only conjecture how long these patients might have survived if their cardiac reserve had been preserved by proper treatment of the anomaly at an early age. Shapiro and Keys[28] reviewed the literature and found that 80% of the patients with patent ductus arteriosus eventually succumb to the cardiac lesion. Patients who were alive at 17 years had an average age at death of 35 years. Deaths in 40% of cases were from endocarditis and endarteritis; in the rest from cardiac failure.

The majority of the authors have agreed with Gross[11] who stated that patent ductus arteriosus should be corrected because [1] physical growth is retarded by the shunt, [2] if the shunt is large, a considerable volume of useless work is performed with a consequent reduction of the cardiac reserve, [3] the hazard of the development of Streptococcus viridans endarteritis exists, and [4] aneurysmal dilatation or embolic phenomena may occur.

When the patent ductus should be closed surgically is still a question. The survey by Waterman, Samson, and Bailey[29] suggested that the preferred ages for this procedure were between 3 and 4 years and between 15 and 20 years. Ziegler,[30] Adams and associates,[31] Hara and co-workers,[32] Ash and Fischer,[33] Clatworthy and McDonald,[34] Rudolph and associates,[35] and Krovetz and Warden[36] recommended early operation because [1] the diagnosis can be made at an early age, [2] the mortality in the unoperated infant is high while the operative mortality is low, despite the seriousness of the condition in these patients, [3] an improvement in growth will be noted and cardiac enlargement and failure, or both, can be prevented, and [4] the incidence of spontaneous closure is low.

Rudolph and co-workers,[35] reviewing Gross's surgically treated series of more than 1,000 patients, were impressed with the group who had shunts of sufficient size to result in embarrassment during the first year of life. Of 21 patients treated surgically, 18 recovered. Three of the 21 had a continuous murmur, but usually the murmur was systolic with maximum intensity along the lower left sternal border similar to ventricular septal defect. The correct diagnosis was suggested when an early diastolic component was heard. A full bounding pulse with a wide pulse pressure aided diagnosis. These patients were studied with retrograde aortography and catheterization, or both. The incidence of associated defects was high. The large volume of flow through the pulmonary artery may make the foramen ovale patent and result in an atrial shunt as well.

Krovetz and Warden[36] recently reviewed the University of Minnesota experience (515 patients). In 435 of these patients, the patent ductus was the only significant lesion. The incidence of congestive heart failure was 5.5% in the entire group and 38% in patients diagnosed before the age of 1 year. In patients over the age of 20 years, the incidence of cardiac failure was 13%. Forty-four percent of the patients were found to have a murmur on visiting a well baby clinic. Special studies were required to confirm the diagnosis or to evaluate suspected pulmonary hypertension in 65% of the patients under 1 year of age. Only 18% of the patients over 1 year of age needed special studies. Cardiac catheterization was performed in 76 patients, and this procedure established the correct diagnosis in 83%. Twenty-one patients had retrograde aortography, which resulted in the correct diagnosis for 86%.

The presence of a second major cardiac lesion led to a poorer prognosis. It was recommended that subacute endarteritis be treated before surgery. In their most recent group, the mortalities occurred in those who had a reversal of flow. In this group, 29% were under the age of 3 years. Now operation is recommended as soon as the diagnosis can be made definitive.

MATERIAL

At the Wilford Hall USAF Hospital 101 patients underwent surgery; their ages ranged from 2 weeks to 38 years. Nineteen of these patients were less than 24 months of age and 15 were

over 16 years of age; 68 of our patients were females and 33 were males. There were 3 deaths.

CASE REPORTS

Case 1. A 5-month-old boy was born prematurely with a birth weight of 1.56 kg. At 3 weeks of age, he went into congestive heart failure which was not controlled with digitalis. Cardiac catheterization revealed a ventricular septal defect, a patent ductus arteriosus, and pulmonary hypertension. At operation the patent ductus was ligated in an effort to eliminate the extra load of this shunt and aid survival until he attained sufficient size and age to withstand a complete corrective procedure. However, three days after operation he died of congestive heart failure. It was presumed that his death was due to the ventricular septal defect.

Case 2. A 21-year-old female presented with mild dyspnea on exertion. She had a large ductus with aneurysmal dilatation of the pulmonary artery. Multiple calcified nodes surrounded the ductus and the pulmonary artery. During the dissection, a rent was made in the pulmonary artery but this was controlled. After the divided ductus had been sutured, the aorta split at right angles to the suture line. Control was difficult and after two more aortic rents were sutured, the heart arrested. This death was considered a technical error.

Case 3. A 2-week-old male was in congestive heart failure with a ventricular septal defect and a patent ductus. He was turned into the lateral position and the endotracheal tube fell out. Anoxic cardiac arrest ensued and resuscitation was ineffective. The death was attributed to the anesthetic technique.

Nine complications were encountered in this series of 101 patients (Table 1). None were serious or significantly prolonged hospital stay.

METHOD

All methods of approach have been used. The ductus has been approached transpericardially and extrapericardially using an anterior thoracotomy incision. Dr. Monro's sternal splitting incision for exposure of the simple patent ductus was not attempted, although this approach is used frequently for incidental ligation of a patent ductus at the time of open-heart surgery. The lateral approach provides much better exposure and it is the one which we recommend, particularly in adults, because more complete dissection of the aorta is in order. The extrapleural approach has been used, especially if open-heart surgery for another defect is contemplated in the future. Although we prefer to divide the ductus, and this procedure is almost always as easy as adequate ligation, the results of multiple ligation have satisfied us fairly well provided that the ductus is small and the patient is in the young age group. Our experience has included only one proved recurrence. This was in an adult who was 35 years of age and had an extremely large ductus. It was thought at the time of operation that division of this ductus would be fraught with danger. Consequently, ligation was accomplished using umbilical tape, and the knot in the umbilical tape was also sutured to prevent loosening. The patient continued to have a murmur, and postoperative catheterization revealed the persistence of the shunt. Not many of our patients have been

TABLE 1
COMPLICATIONS

Chylothorax	1
Wound infection	1
Hemothorax	1
Cardiac arrest	1
Angiospasm, cerebral	1
Schizophrenic reaction	1
Atelectasis	2
Atelectasis, laryngeal edema after bronchoscopy requiring tracheostomy	1
Total	9

TABLE 2
PRESENTING SYMPTOMS

Heart failure	13
Frequent respiratory infection	10
Incidental murmur	77
Subacute bacterial endarteritis	1

TABLE 3
ASSOCIATED DEFECTS

Cardiovascular	
Ventricular septal defect	7
Atrial septal defect	1
Right aortic arch	1
Coarctation	1
Total	10
Urological	
Hypospadias	1
Horseshoe kidney	1
Double left pelvis and ureter	1
Total	3
Eye	
Strabismus	1
Cataracts, nystagmus, and microcephaly	1
Total	2
Miscellaneous	
Short leg	1
Scalp hemangiomas	1
Bilateral inguinal hernia	1
Syndactyly, feet	1
Total	4

catheterized postoperatively but all have been examined six weeks after discharge. With a relatively captive group of patients, it is unlikely that many recurrences have escaped detection.

AGE, DIAGNOSIS, AND DEFECTS

No one has been refused operation because of age. At present it is believed that a patent ductus should be corrected surgically as soon as the lesion can be diagnosed. Thirteen of our patients presented because of heart failure, 10 because of frequent respiratory disease, and 1 because of subacute bacterial endarteritis. The remainder were diagnosed because an incidental murmur was heard (see Table 2).

The diagnosis was made clinically in 76 of the 101 patients. The other 25 were catheterized in order to substantiate the diagnosis. The indications for catheterization were [1] the possibility of an additional defect and [2] the question of pulmonary hypertension. One patient diagnosed clinically was found to have a systemic pulmonary artery connection and one patient had a right-sided ductus. None operated for patent ductus were found to have the ductus not patent at surgery.

Associated defects (Table 3) were present in 19 patients. The system involved most often with an associated defect was the cardiovascular system. Ventricular septal defect was the anomaly most frequently associated with the presence of a patent ductus arteriosus.

SUMMARY

The literature on the surgical treatment of patent ductus arteriosus has been reviewed. Our experience with 101 consecutive cases has been presented. Surgical correction is safe and effective, and it can be accomplished whenever the diagnosis is made.

Presented at the VIII International Congress on Diseases of the Chest, Mexico City, D.F., October 1964.

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The 1967 Influenza B in the USSR

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In January 1967, epidemic outbreaks of influenza B occurred in Moscow and some other towns of the Soviet Union. This is the third epidemic wave of influenza predicted by the USSR Regional Influenza Center. The two previous epidemics occurred in 1962 and 1965, the first being caused by viruses A and B and the second by influenza A2 virus.

DEVELOPMENT OF EPIDEMIC OUTBREAK

From the comparison of data on influenza and some other acute respiratory diseases obtained in 1966 and interepidemic 1964, it would appear certain that the influenza incidence started increasing at the beginning of November 1966. In Dushanbe, Tbilisi, Alma-Ata, and Riga, the number of cases increased by 60 to 65%.

During the first ten days of November 1966, a 30 to 55% increase in acute respiratory disease and influenza incidence was recorded in Baku, Dushanbe, and Khabarovsk, as compared with the same period of 1965. In December 1966, influenza incidence increased in other towns.

In Moscow, a marked increase in incidence was recorded on Dec. 26 and 27 when the number of influenza cases was twice that of the previous days. From the very beginning of January, influenza incidence started increasing sharply and reached its maximum on Jan. 9. Since then, the daily incidence of influenza morbidity commenced declining (see the figure) and continued to do so by the time this communication was written (Jan. 16, 1967).

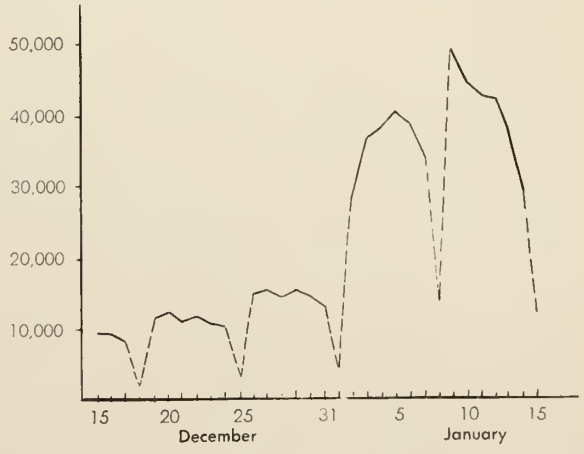
ETIOLOGY OF INFLUENZA

Serologically, the overwhelming majority of influenza cases both in Moscow and in other towns was diagnosed as induced by influenza B virus. In Moscow, the first strain of influenza B virus was isolated in November 1966 when the disease was of a sporadic character. This strain was not antigenically different from the reference strain B/Johannesburg 33/58. After the rise of the epidemic wave, there was isolated a considerable number of strains of influenza B virus which are thoroughly studied at present. According to preliminary data, they are not significantly different in their antigenic structure from the influenza B viruses that caused the 1962 epidemic in this country. However, sera against Johannesburg strain neutralize them only up to the titer of 0.25. In some children's institutions, a few strains of influenza A2 virus were isolated. Thus, one may suggest that the present epidemic wave was caused by influenza B virus with some influenza A2 virus-induced focal episodes and small outbreaks (mainly among children) interspersed.

PROGNOSIS OF EPIDEMIC

The USSR Regional Influenza Center considers prognosis of an influenza epidemic to be one of its main tasks. The possibility of such prognoses is insured by the fact that Moscow Center is supported by a network of some dozens of virological laboratories disposed throughout the country. The presence of this network permits isolation of viruses in different regions and study of the population for the level of immunity.

By the fall of 1966, the Regional Center had the following data available. The last outbreak of influenza B in the USSR was recorded in 1962.



Daily incidence of influenza in Moscow

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The paper was submitted through the Novosti Press Agency.

In the fall of 1965 and at the beginning of 1966, an epidemic of influenza B was registered in many countries of Europe and America. Early in 1966 an insignificant increase in influenza incidence occurred in some towns of the USSR which also was attributed to influenza B virus; however, this rise was not followed by an epidemic and in March the number of influenza cases subsided. Serological examination showed a low level of antibodies in the population.

Thus, influenza B viruses circulated among the population with a low level of immunity against them. The above fact permitted us to put forward a supposition about the possible appearance of an epidemic influenza wave in the autumn or winter of 1966-67.

MEANS OF CONTROL

At present, influenza control aims at providing patients with therapeutic assistance and prevention of complications as well as at the reduction of intensity of the epidemic wave in some communities, since, for the time being, our Public Health Services do not have means available for prevention of an influenza epidemic or arrest of its development in the whole country.

Therefore, the main task of medical institutions during an influenza epidemic is the sound organization of medical assistance among the population. Polyclinics reorganize their work during this period so as to give medical help to most patients with influenza at their homes and prevent the dissemination of the disease through medical institutions. For lack of medical personnel in polyclinics, physicians from other medical institutions and six-year students of medical faculties are engaged in this work. Medical personnel wear masks of cotton wool and gauze with a view to prophylaxis. Medical personnel are alerted to possible influenza complications caused by secondary bacterial infection, for which purpose common antibiotics are used. During the period of an epidemic outbreak, relatives are not permitted to visit patients at general hospitals. Every morning children in crèches and kindergartens undergo medical examination and thermometry in order not to admit those with influenza to healthy groups. Instructive medical work is conducted among the population in newspapers and broadcasting.

SCIENTIFIC INVESTIGATIONS

One of the directions of research in the network of USSR scientific research institutes is the search for influenza vaccines, live and killed. Live and killed vaccines are prepared from the attenuated influenza viruses grown in chick embryo chorioallantoic membrane. Despite efforts in this field, influenza vaccines have been still used on a small scale. The main obstacles impeding their broad application into practice are the rapid alteration of the antigenic structure of influenza viruses (particularly, influenza A viruses), the genetic instability of vaccinal strains, and the lack of genetic laboratory markers which would have corresponded to certain immunological properties of the strains and their residual virulence. For three years, Ivanovsky Institute of Virology has conducted large-scale field tests of live influenza vaccine in Smolensk. Effectiveness in the latest outbreak is being evaluated.

The second direction of scientific investigations, the search for chemotherapeutic means against influenza, is at the initial stage of its development, and for the time being there has not been achieved anything essentially new. Amant adine was not used during the latest epidemic since it affects influenza A virus but it is ineffective with respect to influenza B virus.

The study of interferon is conducted in two directions: [1] interferon production in human leukocytes and [2] induction of endogenous interferon by viral preparations. Both methods of interferon application were being tested during the latest influenza epidemic.

Contradictory references have been made to influenza sera prepared by means of immunization of horses and used for treatment and prophylaxis as a powder administered into the upper respiratory tract. Their effect is of short duration and could cause possible development of allergic reactions. The application of human gamma globulins containing antibodies to influenza virus is more promising for therapeutic treatment, especially as it concerns early therapy of children who may contract influenza.

The problem of influenza control has turned out to be a difficult and complicated scientific research task which is far from being solved.

Each new influenza epidemic is a subject of extensive study and there is a period when new and modified old methods of influenza prophylaxis and treatment undergo a broad practical test. Though progress in this field is slow and not always appreciable, we are prone to evaluate optimistically the perspectives of influenza control in the distant future. The approach in this distant future will depend considerably upon international cooperation in studying influenza as already organized within the framework of the World Health Organization, with its World Influenza Center in London and a network of reference laboratories, one of which is under the direction of the author of this paper.

Karl W. Anderson, M.D.

EARL T. OPSTAD, M.D.
Minneapolis



SCORES OF PEOPLE could claim to have seen Karl Anderson walking in downtown Minneapolis on any given business day, and all of them would unwittingly be guilty of gross understatement.

Karl Anderson has never really been known to walk. He runs. He has been running for half a century and, as anyone who tries to keep pace with him on the sidewalk will attest, only the endless stream of greetings from passing friends breaks Karl's stride enough to save the day for ordinary breathless mortals.

Karl Anderson, in reality, embarked on a career in medicine a-running. The youngest of 7 sons in a family of 10 children, born of Swedish immigrant parents, young Karl was full of ambition and determination and had plenty of opportunity to prove it. His father, a cement contractor, won fame as "Sidewalk Anderson" in an era when wooden sidewalks were being replaced with concrete. The youth found that working for his father during school vacations was invigorating, to say the least.

When the time came for Karl to enroll at the university, his older brothers more or less predetermined that he should become a football player—this was in the days of Henry Williams' famed grid teams. Karl, so thin and lanky that he was nicknamed "Telephone Pole," walked (or ran) each day from the family home at 2508 11th Avenue South to the university.

On that first day of football practice the coach ordered all members of the squad to run 100 yards. The fact that Karl finished far out in front of the others impressed Len Frank, the freshman football coach, who also happened to be the track coach. He gave Karl a football, told him to kick it, then run down field and catch it himself.

That did it. The coach began a quick conversion of the lanky Swede from a potential football player to an assured track star. "These giants of the North would kill you in the first tackle," he said by way of consolation. But the disappointment in the Anderson family was beyond calculation. It was debated whether Karl should even continue his education, and the question was resolved only after the most earnest counsel and discussion.

Some weeks and quite a few track sessions later, Karl became vaguely aware that he was being "sold' on the virtues of a career in medicine. At that time, all athletic trainers were freshman and sophomore medical students. While they were massaging tired muscles, they were also massaging the athlete's brain to think in terms of a medical career. Several of these trainers invited Karl to their medical fraternity for dinner. In due course they accomplished their purpose.

No sooner had he gotten into medical school, however, than his freshman advisor insisted that he give up athletics. "You can't be in medical school and be an athlete too," was the summary edict. Again a family council was called. After all, Karl was captain of the track team that year. It wasn't an honor that he could lightly throw aside and let his teammates down. The decision came largely from the collective judg-

EARL T. OPSTAD *is medical director of Northwestern National Life Insurance Company, Minneapolis.*

ment of his 6 older brothers. "Karl, do the very best you ean in your studies, but eontinue on the traek team," they advised.

Fortunately, Karl had sympathetic understanding in high places. Drs. Riehard E. Seammon and Clarenee M. Jackson, professors of anatomy, and Dr. Elexious T. Bell, professor of pathology, all encouraged him to stay in medical sehool, but to engage in athleties purely for enjoyment and not be distracted by eoaehing offers. In their wisdom they probably knew that to oppose Karl's traek eareer would have been futile, for he went on to compete in the Melrose Games at Madison Square Garden (it was brand-new then and is now soon to be torn down. The Melrose Games are still an annual spring event at the Garden.) There he set a new world's record for the 70-yard high hurdles.

He went on to set still another world reeord in Boston the following night, but in the process broke his ankle and was sidelined until five weeks before the 1924 Olympie tryouts.

He went on to qualify for the Olympics and competed for the United States in Paris in 1924, losing out by a hcartbreaking slip on a wet traek to a man who had never before beaten him.

He went on just one week later, to another world's reeord in Wimbledon Stadium in London, following whieh he was personally entertained by the Duke of York, who subsequently beeame King George VI.

And he went on to earn that cherished medical degree, with such distinetion that at the age of 25 he was already performing major surgery.

Then fate intervened to change his life beyond imagination. Karl W. Anderson, M.D., suffered a subaraehnoid hemorrhage which resulted in total paralysis of his right side and a complete loss of speeeh.

After a year during which he aehieved partial recovery, his restless nature would not permit him to remain idle any longer. It was then that the understanding and wise counsel of Dr. S. Marx White, professor of medicinc at the university, came into play. He took Dr. Anderson under his wing in the clinies at the University Hospital, arranged for him to undertake a three-year fellowship in internal medicine, and instilled in him the confidence to work with the patients despite the severe limitations imposed by his disability. Dr. Anderson became forever grateful for Dr. White's almost parental eoneern at this tragie and uneertain period of his life.

At about this time, Dr. White reeommended to the board of regents the appointment of Dr. Hilding Berglund as the first full-time professor of medicine at the University of Minnesota. Berglund, an aeknowledged genius from Stoekholm, was a highly controversial but most stimulating professor. Karl Anderson was Berglund's first Fellow. He worked under him three years during his fellowship and remained on the staff of the medieal school for two more, advaneing to associate professor of elinieal medicine. Then he embarked on a new career, this time in insurance medieine, when he aceepted an offer as assistant medical director of Northwestern National Life Insuranee Company. He has, however, continued to serve through the years as associate professor of elinieal medieine at the university and expects to retire from this long and happy assoeiation in 1968.

"Some people simply eouldn't take Berglund," Dr. Anderson still recalls, "but I understood his outbursts of anger and his desire for perfeetion. We two Swedes got along beautifully." Many years later Dr. and Mrs. Anderson, on a visit to Sweden, called on the elderly Berglund in Stoekholm, to which he had long sinee returned, and the old gentleman was deeply touehed by the reunion.

Dr. Anderson undertook his duties at Northwestern National Life with eharaeteristie vigor. "I extended my praetiee over the entire United States," he recalls philosophieally. What might otherwise have beeome a routine job of examination and medieal evaluation of insuranee



KARL W. ANDERSON
U.S. Olympic contender and 1924 World Champion, 70-meter high hurdles

applicants, became a many-sided challenge to the intellect of the man who was denied one great medical career but determined to build another.

One of his earliest major achievements was a reevaluation of so-called cardiac invalids—life insurance policyowners who were regarded as totally and permanently disabled by reason of heart attacks they had experienced, and who were drawing disability income from their insurance policies. Dr. Anderson's investigation determined that most of these people were desperately unhappy in their idleness.

Convinced that most of them were not really permanently disabled, and that they would prefer to resume jobs and be useful citizens, he persuaded his management to offer this proposition: Every such cardiac patient was encouraged to seek employment. The company would continue to pay the full amount of monthly disability income, regardless of how much salary or other income the person began earning. Then at the end of one year that person would have the choice of remaining employed and giving up the disability income, or going back to idleness.

About 80% of these people accepted the offer, again found happiness in daily work, and in due course went off disability income. This astonishing experiment spread through the insurance industry from coast to coast. The companies obviously saved money, and one can reasonably assume that thousands of heart patients again found enjoyment and satisfaction in their daily lives.

In 1933 Dr. Anderson presented his first professional paper. It was delivered to members of the Milwaukee Academy of Medicine and conveyed an optimistic viewpoint toward cancer of the stomach. Dr. Anderson was acquainted with the then-revolutinary theory of Dr. Owen Wangensteen that patients with stomach cancer, hitherto thought to be inoperable, might be saved through radical resection. He referred four such cases to Dr. Wangensteen, who operated on and saved all of them, and these histories were related in Dr. Anderson's paper.

In 1937 Dr. Anderson noted studies which demonstrated that nicotine was transferred by the smoking mother to her baby via the mother's milk. The results of these studies, publicized by Northwestern National Life, resulted in vigorous protests by the major tobacco companies. Their representatives personally requested the company's president, Mr. O. J. Arnold, to issue a retraction. Mr. Arnold called in Dr. Anderson to present the substantiating data, and promptly let it be known that no retraction would be issued in the face of the factual evidence thus documented. This represented one of the very first steps in what now, thirty years later, has become a major campaign to alert the public to the dangers of cigarette smoking.

With the passing of time, Dr. Anderson perhaps became best known within Northwestern National Life for his espousal of geriatrics, which he popularly referred to as "The science of helping older people enjoy life longer." Starting in 1946, when wartime experience had made it obvious that antibiotics were going to result in a lengthened life span for many people, he embarked on a series of pamphlets, ultimately 19 in number, presenting a reassuring picture based on the latest developments in the treatment of common ailments of later life. Bearing such catchy titles as, "Taking the Die Out of Diabetes," "Are You the Ulcer Type?" "Fat Can Be Fatal," and the like, they immediately proved popular. Requests for copies continue to come in a steady flow to this day.

Inevitably, Dr. Anderson's professional competence would be recognized nationally in the life insurance industry. He became a diplomate of the Board of Life Insurance Medicine. He is a past chairman of the medical section of the American Life Convention, one of the industry's top management associations, and in 1954 won wide acclaim for an address before this group entitled, "Breaking the Age Barrier." In this he projected the problems which society would eventually face as steadily increasing longevity collided with legislated old-age idleness. Dr. Anderson also holds membership in the American Medical Association.

It is at the local and state level, however, where his professional, civic, and charitable activities have marked him as a veritable dynamo—and where he has formed his strongest friendships and found deep personal satisfaction. He is a member of the Minnesota Society of Internal Medicine and the Minnesota Pathological Society. He is a past chairman of the Health and Medical Care Division of the United Fund of Hennepin County, has served on the board of directors of the Hennepin County Red Cross for more than a decade, and has long been active in the Twin City Diabetes Association.

He is a past president of the Minnesota Heart Association, past president of the Minneapolis Academy of Medicine, current president of the Minnesota Medical Foundation, and also was treasurer of the Minnesota State Medical Association for ten years, in which capacity he enabled

that body to attain an outstanding record of fiscal accomplishment. He is also a trustee of Swedish Hospital. Notwithstanding this array of interests, he found time to serve for many years as a trustee of Bethlehem Baptist Church, in which capacity he provided invaluable financial counsel to that long-established congregation.

In October 1965 Dr. Anderson was honored by the University's Alumni Association with the Harold S. Diehl Award, and with it a gold medal, for outstanding service to the medical school, the medical profession, and the University of Minnesota. More recently he received the Distinguished Service Award of the Minnesota State Medical Association, also signified by a gold medal.

His retirement as Medical Director of Northwestern National Life Insurance Company became official early in 1966, but it has been only nominal retirement. It merely means that he is freer to spread his enthusiasm and optimism into more areas than before. And in busying himself in those areas, he is harder than ever to reach on the telephone.

Karl Anderson is not one to lack a proper appreciation of the past, nor the value of friendship, loyalty, a charitable viewpoint, and a helping hand when most needed. He has an abiding interest and faith in the potential of youth. The fact that he has continued over the years to keep in touch with Phi Beta Phi, his medical fraternity; the Incus Society, honorary medical fraternity; and the Gray Friars, undergraduate honorary medical fraternity, inevitably leads one to ask: Does the great runner of yesteryear still maintain an interest in athletics and specifically in the Olympics? Indeed he does!

On the current letterhead of Olympian International, a global association of yesterday's amateur athletic greats, is the name of Karl W. Anderson, vice-president of the Midwest chapter. He and Mrs. Anderson attended the Olympic Games in Rome in 1960, where Dr. Anderson was a member of the honor guard. Currently they have their eye on Mexico City and the 1968 Olympics. Dr. Anderson will not be competing on the track there, but if he were, he would be in top condition and a favorite to win. He has been over a thousand hurdles since Paris in 1924 and has crossed all of them with room to spare.

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Book Reviews . . .

Advances in Anesthesiology: Muscle Relaxants

Edited by LESTER C. MARK, M.D., *and* E. N. PAPPER, M.D., *128 pp., ill., Hoeber Medical Division, Harper & Row, New York City, 1967. $6.75*

The editors tell us in their introduction that the work is an outgrowth of a postgraduate course presented in December 1965 by the department of anesthesiology of the College of Physicians and Surgeons at Columbia University. And they suggest they may offer additional volumes as suitable material gathers from the departmental research program.

The book consists of 7 succinct chapters by as many authors. Physiology, chemistry, pharmacology, and genetic aspects of neuromuscular transmission are discussed in 4 chapters and the remaining 3 deal with the mechanisms of skeletal muscle relaxation, the clinical use of relaxants, and myasthenia gravis. As might be expected when each author has responsibility for only a single chapter, the book is virtually free of error with correct, clear illustrations and nicely polished prose.

At times, however, the polish dazzles us unduly as when, in the midst of a description of the termination of acetylcholine's activity, we read "The imidazole nitrogen strongly attracts the hydrogen of the serine hydroxyl, rendering the hydroxyl oxygen relatively more negative and, hence, a better nucleophile. The serine hydroxyl oxygen is now in position to mount a nucleophilic attack on the polarized carbonyl carbon atom of acetylcholine." But such heady stuff is rare and, on the whole, the authors maintain an equitable division between the academic and the clinical. Each chapter closes with a useful summary and is followed by an adequate, current list of references.

The reader looking for a complete book on muscle relaxants will not find it here. But he will be rewarded with much that is new and exciting in the field. The subject matter will interest all students of anesthesiology and many of neurology, internal medicine, biochemistry, pharmacology, and physiology.

J. R. GORDON, M.D.
Minneapolis

Chemotherapy of Disseminated Solid Tumors

by FRED J. ANSFIELD, M.D., *Charles C Thomas Co., Springfield, Ill., 1966. $7.50*

The personal experience of the author, in treating over 3,000 patients with disseminated solid tumors, is reflected in this publication. Only a limited number of references are employed in the publication, since the statements in the text are primarily based upon the author's own observations, impressions, and conclusions. The author has had a broad experience with the fluorinated pyrimidines. This experience is evident in the weighting of the book with work with these agents.

The treatment of disseminated solid tumors is described, particularly emphasizing cancers of the breast, gastrointestinal tract, lung, and testes. Methods of drug evaluation and management of drug toxicity are described, but the latter is confined to a few agents. In many instances, the treatment of specific tumors is brief and incomplete. It fails to provide the data that would be necessary for a physician caring for these types of lesions.

Though this treatise was prepared as a reference to the practicing physician, it fails to supply the necessary details for the inexperienced physician to meet the needs of patient care. It has some very interesting sections of cancer chemotherapy which particularly reflect the broad experience of the investigators. Unfortunately, the attempt to provide a ready reference for the management of chemotherapy has not been attained in this publication. There is no inclusion of the chemotherapy of hematological neoplasms which are an essential part of the new field of oncology. This does represent, however, one of the beginning efforts of oncologists to make available to the practicing physician information regarding the care of the patient with advanced malignant disease.

B. J. KENNEDY, M.D.
Minneapolis

One Hundred Years of Medical Progress: A History of the New York Medical College Flower and Fifth Avenue Hospitals

by LEONARD PAUL WERSHUB, M.D., *259 pp., ill., Charles C Thomas, Springfield, Ill., 1967. $6.50*

No one interested in the rise and development of American hospitals can fail to appreciate this organizational history of a small homeopathic hospital which became a distinguished medical center. Chartered in 1860, The Homeopathic Medical College of the State of New York in New York City altered its corporate name 4 times before 1938 when it became the New York Medical College, Flower and Fifth Avenue Hospitals. Dr. Wershub's compact and tightly written book relates the events of the institution's growth during the seventy-eight years between the time it was chartered and 1938. But this is not all. He sketches the formative years before 1860 and pays attention to the decades following 1938.

The first 3 chapters lay the background, stress the state of homeopathic medicine, indicate the support of William Cullen Bryant who was "the first in the City of New York to accept homeopathy as a new healing system" and later the first president of the college,

(Continued on page 12A)

(Continued from page 11A)

describe the sanitary conditions of the city, and discuss conflicting views of practice held by allopaths and homeopaths.

Dr. Wershub, however, makes it clear that, from the beginning, the college offered instruction in both allopathic and homeopathic systems of medicine. His later chapters, containing the usual story of political and professional bickering, also indicate changes in curriculum. In 1869, for example, a new charter omitted the word "homeopathic." During the decades before 1900, revised charters, mergers, and construction of buildings slowly resulted in what was to become a major institution. Although 2 world wars offered vexing problems, the institution weathered them, but not without internal difficulties. Finally, in 1955, the New York Medical College Metropolitan Center was created. What had begun as a college with 2 rooms was now a thoroughly modern teaching institution associated with 3 hospitals with more than 3,000 beds. The 1960s were to witness additional growth.

Writing with justifiable pride, the author says: "As we approach the second hundred years . . . we are the sixteenth largest of the nation's medical schools . . .; that through its voluntary hospitals and affiliations with municipal hospitals medical care is provided for over 2,000,000 people annually . . .; that its medical center includes nearly 3,500 beds for teaching, research, and patient care." If the college is proud of its progress, as it should be, it should also take pride in its history which Dr. Wershub has written.

PHILIP D. JORDAN, PH.D.
Minneapolis

Cardiac Surgery

edited by JOHN C. NORMAN, M.D., *603 pp., ill., Appleton-Century-Crofts, New York, 1967. $15 (cloth) $9.75 (paperback)*

Current and comprehensive information in a field as subject to change as cardiac surgery is rarely found in textbook form. This recently published monograph on cardiac surgery edited by John C. Norman of Harvard Medical School is an exception, for he and his 56 coauthors have covered this rapidly expanding field in a clear, concise, and up-to-date manner. Available in both cloth cover and paperback, the 603 pages of this treatise are introduced with a stimulating foreword by Dr. F. D. Moore. After a brief historical review, the remainder of the material is divided into 5 sections: [1] General, [2] Cardiac Surgery in Infants and Children, [3] Cardiac Surgery in Adults, [4] Complications of Cardiac Surgery, and [5] Recent Developments in Cardiac Surgery.

The various subjects under these broad headings are, for the most part, covered in a readable and understandable fashion, but as in any book with multiple authorship, the lack of consistency is at times distracting. Thus, some chapters are written almost in outline form, whereas others are detailed and unusually exhaustive. Most chapters include a list of references; in some, they are numbered and referred to in the text, while in others they are merely listed alphabetically. Occasionally the list is exhaustive, while in a few chapters the bibliography is skimpy or absent. More careful editing might have eliminated these inconsistencies. For the most part, though, the editor has avoided the usual pitfalls of a multiauthored book and has provided the reader with a text of logical continuity and clarity. The illustrations, charts, and graphs, together with the print, are all designed to pamper the reader.

The rather detailed nature of the monograph will be less appealing to the medical student than to the specialist or to the postgraduate who plans to practice cardiology or cardiac surgery. To anyone at all interested in this fascinating field, *Cardiac Surgery* should serve as a fine reference book.

F. HENRY ELLIS, JR., M.D.
Rochester

Foot Disorders: Medical and Surgical Management

by NICHOLAS J. GIANNESTRAS, M.D., *521 pp., ill., Lea & Febiger, Philadelphia, 1967. $22*

This is a comprehensive book on diagnosis and treatment. It is an excellent reference for the orthopedist and general practitioner. The book is well written and illustrated and contains an excellent bibliography. It not only discusses surgical conditions but covers medical complaints and management as well. The first two chapters dealing with anatomy and physiology are excellent reviews, and the third and fourth chapters answer the questions on "what is normal" as well as normal development and care. This is particularly important to the family physician in the role of preventive care. The author then covers the usual disabilities such as clubfoot, cerebral palsy, polio, and so forth. The discussion on fractures is primarily on closed treatment and is somewhat brief. Over all, the author's selection of topics is excellent and this text should be found in the libraries of most orthopedists and general practitioners. The book is well printed and adequately indexed.

H. J. ERICKSON, M.D.
Minneapolis

International Encyclopedia of Pharmacology and Therapeutics, Section 6, Volumes I and II: Clinical Pharmacology

edited by L. LASAGNA. *V. I, 373 pp., ill., Pergamon Press, Inc., Long Island City, New York, 1966. $12.50. V. II, 667 pp., ill., Pergamon Press, Inc., Long Island City, New York, 1966. $9.50*

Recent medicopolitico events have thrust clinical pharmacology and the ethics of human drug evaluation into unaccustomed prominence. The podium of public opinion has demanded that those professionals, skilled in the objective assessment of therapeutic response (for example, clinical pharmacologist), provide a responsible liaison between clinical therapeutic reality and the phantasmagoria of pharmaceutical advertising.

Does this require an omniscient and omnipotent being? While such powers would undoubtedly prove useful, it is well to remember that the clinical pharmacologist is "mere mortal" and represents a unique synthesis between the clinically skilled physician and the scientifically disciplined pharmacologist. In a general

(Continued on page 17A)



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Season

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this issue: smog, smaze or smust...

Smog, smaze or smust...effects of air pollution on upper respiratory tract

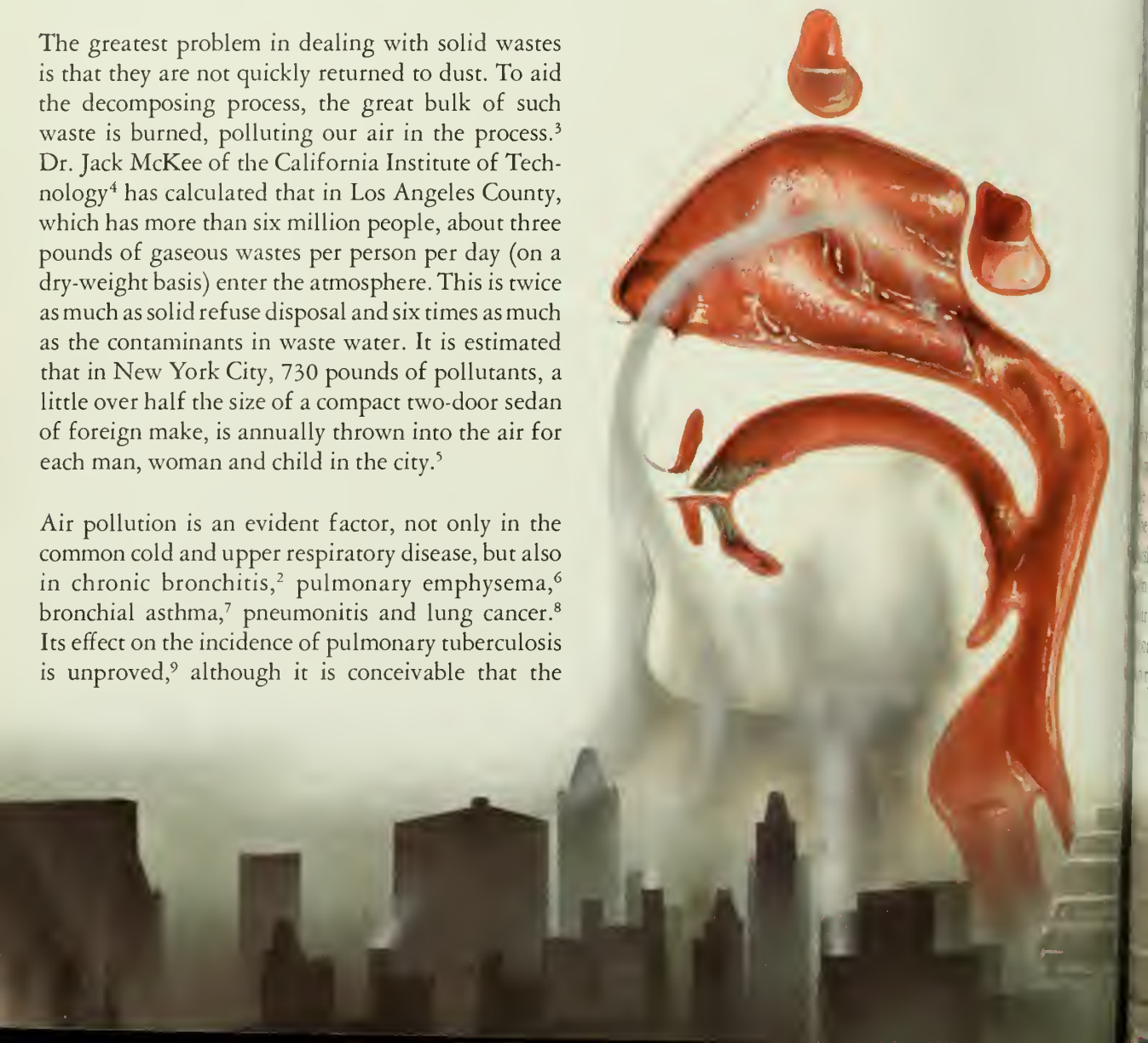
Nathan Flaxman, M.D., Diplomate, American Board of Internal Medicine, Chicago, Illinois

In Los Angeles it is *smog* (smoke and fog). In New York City *smaze* (smoke and haze). In El Paso *smust* (smoke and dust). The original factor was smoke plus such natural phenomena as fog, haze and dust, but air pollution has mushroomed from a smoke problem in our industrial cities into a major economic, esthetic and public health problem that affects practically every American locality and citizen.[1,2] Respiratory disease, of course, is by far the most costly effect of air pollution, for contaminated air can aggravate our illnesses, deplete our strength and shorten our life span.[1]

The greatest problem in dealing with solid wastes is that they are not quickly returned to dust. To aid the decomposing process, the great bulk of such waste is burned, polluting our air in the process.[3] Dr. Jack McKee of the California Institute of Technology[4] has calculated that in Los Angeles County, which has more than six million people, about three pounds of gaseous wastes per person per day (on a dry-weight basis) enter the atmosphere. This is twice as much as solid refuse disposal and six times as much as the contaminants in waste water. It is estimated that in New York City, 730 pounds of pollutants, a little over half the size of a compact two-door sedan of foreign make, is annually thrown into the air for each man, woman and child in the city.[5]

Air pollution is an evident factor, not only in the common cold and upper respiratory disease, but also in chronic bronchitis,[2] pulmonary emphysema,[6] bronchial asthma,[7] pneumonitis and lung cancer.[8] Its effect on the incidence of pulmonary tuberculosis is unproved,[9] although it is conceivable that the presence of various materials polluting the air might do this. A siege of smog in Denver, the "mile high city," in December 1965 was accompanied by respiratory infection that doubled normal absentee rates in schools, factories and city government.[10]

While air pollution is only one factor, it has become important in the causes of most of the afflictions of the respiratory tract. This has been shown not only by the Denver occurrence, but also by detailed study[2] of respiratory illness in a small group of 313 men



from October 1962 to May 1963 when there were 202 episodes involving the upper respiratory tract. The attack rate of illness was related in time to increased concentration of both smoke and sulphur dioxide in the atmosphere of the district in which the men lived.

Other factors often mentioned, include exposure to those who have colds, exposure to extreme changes of temperature, allergy and bacterial infection. However, when low individual resistance due to lack of rest, overwork, fatigue, improper or unbalanced diet, previous illness and emotional stress are included as causes, we enter the realm of somewhat obscure relationships. Much more emphasis can be placed on the role of polluted air.

The symptoms, signs and complications of involvement of the upper respiratory tract, especially the common cold, are the same regardless of the causative factor. Swelling of the lining of the nose, the scratchy dry throat, the discharge from the nose at first watery then thicker, discolored and more tenacious, the eyes tearing, and frequent sneezing are all part of the Number 1 human ailment. Concurrent or residual sinusitis when mucus is trapped there, middle ear involvement due to interference with drainage, laryngitis and bronchitis are complications of the common cold. The primary interference is with a most important function of the nose—the cleansing of foreign matter in the first line of "air defense" to prevent it from entering the breathing tract.

However, the diagnosis and subsequent decision on how to treat the patient so affected rests basically on the relief of symptoms that cause him the misery. The stuffed, runny nose, the clogged ears, and the harsh dry cough—all the symptoms that make common cold sufferers feel miserable and interfere with their sleep—can be alleviated with medications of the oral nasal decongestant/antihistamine combination type. The burning sensation in the throat, soreness of the chest and even chest pain can also be relieved by such medication. Rest in bed if there is fever (but confined to home at least), liberal fluids, uniformly warm surroundings and adequate humidity in the room, are all helpful adjuncts to the medication. Most common cold sufferers recover rapidly and are symptom-free in four to ten days.

Further treatment, altered by the fact that the affliction hangs on for more than the usual duration of the common cold, requires consideration of allergy, which is most frequently the prolonging factor. But air pollution itself may often be the culprit.

(Concluded on following page)

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Phenylpropanolamine hydrochloride	12.5 mg.
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At the Third National Conference on Air Pollution held recently, it was emphasized that this subject had received more attention in the past four years than in all previous history. Spicer,[11] an active participant at this conference, reiterated that it behooves the practicing physician to be aware of trends in respiratory disease and to accept a major role in community action relating to air pollution and respiratory health. By taking a positive stand physicians have been instrumental in the development of anti-pollution legislation. An outstanding example is Los Angeles where major steps have been taken by abolishing coal burning, and even banishing oil burning, seven months a year. Natural gas must be used instead and it must be used by industry when available. Backyard incinerators have been abolished in favor of landfill disposal, and building incineration ended except for a few expensive smokeless furnaces.[10] Concerted action can be taken against particular industrial nuisances. One company that disregarded complaints discovered its error when thousands of its credit cards were returned by irate customers who decided to patronize competing companies.[12]

Summary. Respiratory disease is the most important and most costly effect of air pollution, whether termed smog, smaze, or smust. Air pollution is an economic, esthetic and public health problem that affects practically every American locality and citizen. New sources of air pollution are invisible and odorless, but the harmful gases and liquid droplets are there. Triggered by sunlight, some of these undergo mid-air chemical changes and the results are even more irritating to the upper respiratory tract. The symptoms, signs and complications, especially of the upper respiratory tract, can be readily aborted by modern medication but may be unduly prolonged by polluted air. In steps taken to prevent this, the practicing physician can take a major role.

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(Advertisement)

(Continued from page 12A)

sense, his responsibilities to the medical community are extraordinarily diverse; falling within his province are the detailed evaluation of drugs in man, the development of suitable methodology for analyzing and testing drugs in man, and the utilization of basic pharmacologic data in the establishment of clinically valid therapeutic regimens.

The volumes prepared by the International Union of Pharmacology (Clinical Pharmacology I and II) under the editorial guidance of Dr. Louis Lasagna promise to fulfill a significant role by concisely describing the functions of the clinical pharmacologist and how he may be of help to the clinical practitioner.

The text analyzes, in considerable detail, methods for evaluating the effects of pharmacologic agents upon diverse pathophysiologic states. The format of the book is convenient and well organized in that drug effects are discussed within the context of discrete organ systems or symptom complexes. Consequently, no orientation towards a specific specialty group is apparent; however, any practitioner will find much of value if he is concerned with objectively assessing (qualitatively and quantitatively) the effects of therapeutic procedures in his patients.

A primary omission in this symposia appears to be the absence of any discussion on drug interactions at the clinical level. It is most unfortunate since it poses one of the most vexing and complex problems in contemporary clinical therapeutics.

BERNARD L. MIRKIN, PH.D., M.D.
Minneapolis

NEW BOOKS RECEIVED

Great Ideas in the History of Surgery
by LEO M. ZIMMERMAN, M.D., and ILZA VEITH, Ph.D., 2d rev. ed., 587 pp., ill., Dover Publications, Inc., New York City, 1967. $3 (paper)

Treatment of Hyperthyroidism with Radioactive Iodine: A Twenty-Year Review
by JOHN E. KEARNS, M.D., 73 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $6.50

Intravenous Abdominal Aortography and Placentography
by MELVYN H. SCHREIBER, M.D., FRED J. WOLMA, M.D., and CHARLES K. HENDRICK, M.D., 57 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $3.75

Cardiac Pacemakers
by HAROLD SIDDONS, F.R.C.S., and EDGAR SOWTON, M.D., 331 pp., ill., Charles C. Thomas Co., Springfield, Ill., 1967. $16.75

Fundamentals of Neuropathology
by WILLIAM BROOKS DUBLIN, M.D., 2d ed., 478 pp., ill. Charles C Thomas Co., Springfield, Ill., 1967. $24.50

From One Surgeon's Notebook
by LOYAL DAVIS, M.D., 145 pp., Charles C Thomas Co., Springfield, Ill., 1967. $5.75

Neurological Problems
edited by JERZY CHOROBSKI, 444 pp., ill., Pergamon Press, New York City, 1967. $13.50

Handbook of Orthopedic Surgery
by ALFRED R. SHANDS, JR., M.D., and BEVERLY HANEY, M.D., 7th ed., 572 pp., ill., C. V. Mosby Co., St. Louis, 1967. $12

Atlas of Intestinal Stomas
by RUPERT P. TURNBULL, M.D., and FRANK L. WEAKLEY, M.D., 207 pp., ill., C. V. Mosby Co., St. Louis, 1967. $21.50

(Continued on page 18A)

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(Continued from page 17A)

Ficatul Si Hormonii

by ST.-M. MILCU, L. VAISLER, and EMMA COSTINER, 372 pp., ill. Editura Academiei Republicii Socialiste Romania, Bucharest, 1967.

Color Atlas of External Manifestations of Disease

by G. F. KLOSTERMANN, H. SÜDHOF, and W. TISCHENDORF, 298 pp., ill., The Blakiston Division, McGraw-Hill Book Company, New York City, 1967. $25

Introduction to Scientific Psychiatry: A Behavioristic Approach to Diagnosis and Treatment

by H. A. STORROW, M.D., 256 pp., Appleton-Century-Crofts, New York City, 1967. $6.95



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Ciba Foundation Study Group No. 25: Mongolism

edited by G. E. W. WOLSTENHOLME and RUTH PORTER, 99 pp., ill., Little, Brown and Company, Boston, 1967. $3.50

Ciba Foundation Study Group No. 26: Effects of External Stimuli on Reproduction

edited by G. E. W. WOLSTENHOLME and MAEVE O'CONNOR, 197 pp., ill., Little, Brown and Company, Boston, 1967. $3.50

Care of the Patient with a Stroke

by GENEVIEVE WAPLESS SMITH, R.N., 148 pp., ill., Springer Publishing Co., New York City, 1967. $3.25

Ciba Foundation Symposium: Development of the Lungs

edited by A. V. S. de REUCK, M.Sc., and RUTH PORTER, M.R.C.P., 408 pp., ill., Little, Brown and Company, Boston, 1967. $13

Ciba Foundation Study Group No. 28: Thiamine Deficiency: Biochemical Lesions and Their Significance

edited by G. E. W. WOLSTENHOLME, F.R.C.P., and MAEVE O'CONNOR, B.A., 163 pp., ill., Little, Brown and Company, Boston, 1967. $4.95

Caste and Race: Comparative Approaches: A Ciba Foundation Volume

edited by ANTHONY de REUCK and JULIE KNIGHT, 384 pp., Little, Brown and Company, Boston, 1967. $12

Ciba Foundation Colloquia on Endocrinology: Endocrinology of the Testis: Volume 16

edited by G. E. W. WOLSTENHOLME, F.R.C.P., and MAEVE O'CONNOR, B.A., 331 pp., ill., Little, Brown and Company, Boston, 1967. $12.50

Science and Psychoanalysis: Volume XI: The Ego: Scientific Proceedings of The American Academy of Psychoanalysis

edited by JULES H. MASSERMAN, M.D., 234 pp., Grune & Stratton, New York, 1967. $9.75

Lipid Metabolism in Tissue Culture Cells: The Wistar Institute Symposium Monograph Number 6

edited by GEORGE H. ROTHBLAT and DAVID KRITCHEVSKY, 164 pp., ill., Wistar Institute Press, Philadelphia, 1967. $5

Mathematical Challenges to the Neo-Darwinian Interpretation of Evolution: The Wistar Institute Symposium Monograph Number 5

edited by PAUL S. MOORHEAD and MARTIN M. KAPLAN, 140 pp., ill., The Wistar Institute Press, Philadelphia, 1967. $5

Principles of Head and Neck Surgery

by H. ROBERT FREUND, M.D., 337 pp., ill., Appleton-Century-Crofts, New York, 1967. $12.50

Antibodies to Biologically Active Molecules: Proceedings of the 2nd Meeting of the Federation of European Biochemical Societies, Vienna, 21-24 April, 1965: Volume I

edited by BERNHARD CINADER, 424 pp., ill., Pergamon Press, New York, 1967. $18.50

Prothrombin in Enzymology, Thrombosis and Hemophilia

by WALTER H. SEEGERS, Ph.D., 181 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $9.75

Gaseous Digestive Conditions: Including Belching, Bloating, "Wind" and Air Swallowing. What They Are, What Causes Them and How They Can be Controlled

by JOSEPH H. POLLOCK, M.D., 163 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $7

A Biological Consideration of Treatment of Breast Cancer

by GEORGE CRILE, JR., M.D., 189 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $8

Symposium on Oral Sensation and Perception

compiled and edited by JAMES F. BOSMA, M.D., 360 pp., ill., Charles C Thomas Co., Springfield, Ill., 1967. $18.50

News Briefs . . .

North Dakota

DR. JAY T. RAUH, a native of Maryland, joined the medical staff of the Northwest Clinic at Minot as a specialist in obstetrics-gynecology.

* * * *

DR. JEROME P. HAGER, formerly of Hankinson, joined the staff of the Fargo-Moorhead Psychiatric Center.

* * * *

DR. PERRY ENGSTROM, surgeon with the Red River Valley Clinic, accepted a second two-month tour of duty in Vietnam as a participant in the American Medical Association's program, "Volunteer Physicians for Viet Nam." Dr. Engstrom's first tour, under the same program, was in February and March of 1966.

* * * *

DR. CURTIS L. BAKKEN, formerly of Hawley and Moorhead, Minn., opened a pathology practice in association with Dr. Ralph E. Tarnasky at St. Alexius Hospital, Bismarck. Drs. Bakken and Tarnasky are also pathologists at the Mandan Hospital.

* * * *

RECENT ADDITIONS to the staff of the Public Health Service Indian Hospital at Belcourt are Drs. John H. Muus of Grand Forks, V. James Thompson of Beach, and Carl Adrian Robson of Hinsdale, Ill.

* * * *

A GRANT of $188,010 to the North Dakota Medical Research Foundation was announced by the U.S. Public Health Service. Coordinator for the grant is Dr. Theodore Hardwood, dean of the University of North Dakota Medical School. Purpose of the grant, which is for the first year of a two-year program, is "to improve diagnoses of and treatment of heart disease, cancer, strokes, and related illnesses."

* * * *

DR. H. CHARLES WALKER, formerly of Williston, is now serving the Devils Lake area as consultant in radiology. He is head of the department of radiology at Mercy Hospital, consultant to the Lake Region Clinic, and in charge of the radiology department at the Fargo Veterans Administration Hospital.

* * * *

LICENSED RECENTLY by the State Board of Medical Examiners were the following 16 physicians: Drs. Gary D. Bergman, Minot; Ronald D. Brown, Ellendale; William D. Erickson, Rochester, Minn.; Hector Gallego-Correa, Minneapolis; Ted M. Haley, Grand Forks; Orest E. Horodysky, Dugway, Utah; James R. Householder, Minneapolis; Norman M. Kester, Rolette; Norbert J. O'Keefe, Spring Valley, Minn.; Allen W. Pepple, Richmond, Va.; Jay T. Rauh, Baltimore; Wilson M. Salter, Minneapolis;

(Continued on page 20A)

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References

1. Combes, F. C. and Canizares, O. *New York State J. Med.* 52: 706-708 (Mar. 15) 1952
2. Xander, G. W. *Western Med.* 1: 14 (Sept.) 1960.
3. Baker, A. G. *Penna. Med. J.* 63: 697-698 (May) 1960
4. Lehrer, H. W., Lehrer, H. G., and Lehrer, D. R. *Northwest Med.* 75: 1249-1252 (Nov.) 1955

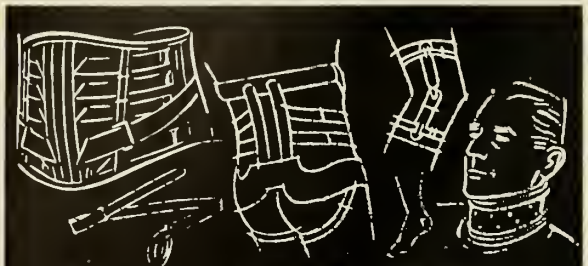
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NEWS BRIEFS

(Continued from page 19A)

Walter G. Schroeder, Grand Forks; Robert W. Spletstoesser, Ashley; John H. Uecker, Grand Forks; and John R. Wasleigh, Tucson, Ariz.

* * * *

ADDITIONS to the staff of the Fargo Clinic are Dr. James R. Householder, ophthalmology; Dr. Arnold Medved, dermatology; and Dr. Thomas L. Sussex, obstetrics-gynecology.

Minnesota

DR. JOHN RAUENHORST, formerly of Slayton, will be on duty for the next two years as a public health service officer assigned to the Peace Corps in Caracas, Venezuela.

* * * *

DR. CHARLES P. KAMPEN, a native of Le Center, joined the Albert Lea Clinic for the summer. He will be stationed in the Philippines with the Air Force, beginning this month.

* * * *

DR. RICHARD J. SNYDER resigned from the Mankato Clinic to begin practice in Modesto, Calif.

* * * *

DR. W. D. BROWN, SR., dean of Minneapolis Negro general practitioners, retired after forty years of practice in his native city. He is a 1925 graduate of the University of Minnesota College of Medicine.

* * * *

DR. WALTER F. LARRABEE, JR., chief of service at Willmar State Hospital, succeeded Dr. Donald C. Carter as acting medical director. Dr. Carter has joined the faculty of the University of Missouri School of Medicine.

* * * *

DR. KENNETH L. BURESH, formerly of Westbrook, has joined the Owatonna Clinic.

* * * *

DR. H. M. FISCH resigned as Austin city health officer after twenty-seven years in the post. He will continue to serve until a successor is appointed.

* * * *

DR. HILDEGARD GRABER of Newport was appointed assistant medical director of Hastings State Hospital.

* * * *

DR. HOWARD ANDERSEN of Rochester, a Mayo Clinic specialist in lung diseases, was named to the 7-member State Pollution Control Agency.

* * * *

DR. ROBERT MULHAUSEN, former assistant chief of medicine at Minneapolis Veterans Hospital, was named assistant dean of the University of Minnesota College of Medical Sciences. He succeeds Dr. N. L. Gault, Jr., who joined the University of Hawaii Medical School as professor of medicine and associate dean for the international program.

NEWS BRIEFS

DR. CHARLES F. TURBAK of Fridley is now associated with Drs. Patrick Meekin, Thomas Meany, and Joseph Stenzel of Minneapolis.

* * * *

DR. JOHN A. DOCHERTY resigned as medical director of Anoka State Hospital to join the staff of Hennepin County General Hospital. Dr. Guido H. Schynoll, assistant medical director at Anoka, was named acting medical director.

* * * *

DR. RAYMOND J. JACKMAN of Rochester was appointed to an new term on the State Board of Health.

* * * *

DR. W. C. FERGUSON, Walnut Grove physician and surgeon for thirty years, discontinued his medical practice because of illness.

* * * *

DR. GERARDO A. CRICHIGNO, a native of Buenos Aires, Argentina, joined the medical staff of Hastings State Hospital.

* * * *

DR. DAVID SWEET, who recently became associated with Drs. E. A. Thayer, O. E. Wandke, and G. N. Kraemer of Fairmont, began two years of active duty as an Air Force medical officer.

DR. PETER A. LOMMEN, JR., of Austin has been reelected to active membership in the American Academy of General Practice.

* * * *

DR. JOE M. NEAL of Edina is retiring from the Veterans Administration after forty-seven years. For the last decade he has been medical consultant in the insurance claim section of the VA center at Fort Snelling.

* * * *

DR. O. LEWIS ZAHRENDT of Golden Valley returned from a 117-member group tour of outstanding medical centers in the Orient.

South Dakota

DR. MILFORD O. ROUSE, president of the American Medical Association, was guest of honor at a 3rd District Medical Society dinner at Brookings.

* * * *

DR. HILARY CONNOR, formerly of Providence, R.I., joined the Aberdeen area office of the Public Health Service as chief of the maternal and child health branch and acting chief of the office of program services.

* * * *

DR. E. W. URBANYI, formerly of Gettysburg, joined the staff of the Veterans Administration Hospital at St. Cloud, Minn.

(Continued on page 22A)

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NEWS BRIEFS

(Continued from page 21A)

MEDICAL STAFF of the new $650,000 Coteau Des Prairies Hospital at Sisseton consists of Drs. Walter Brinkman, Harry Brauer, and Ed Batt. Other physicians are welcome to have patients at the hospital.

* * * *

DR. ALLAN CUNNINGHAM, formerly of Appleton, Wis., replaced Dr. Robert Palmer at the Public Health Service Hospital at Sisseton. Dr. Palmer has entered private practice.

* * * *

DR. JEROME G. KADELL, a gradnate of the University of Wisconsin Medical School, was assigned to the Public Health Service Hospital at Wagner.

* * * *

DR. CURT ROSENBAUM, formerly chief of staff at the Fort Meade Veterans Administration Hospital, was transferred to the Veterans Administration Hospital at Salem, Va.

* * * *

DR. ROMANS AUSKAPS joined the Bartron Clinic, Watertown. He is also medical director of the Memorial Hospital Rehabilitation Center.

Cartoon by Whitney Darrow, Jr.



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NEWS BRIEFS

DR. JAMES EDWARD RYAN of the Spiry Clinic, Mobridge, was elected to active membership in the American Academy of General Practice.

* * * *

DR. JOHN W. ARGABRITE, an associate in the Brown Clinic at Watertown since 1948, resigned to construct a clinic and pharmacy for a medical practice limited to asthma, allergic emphysema, hay fever, and other allergic conditions. The new clinic will be adjacent to the Northwest Bell Telephone Co. building in Watertown.

* * * *

DR. JAMES DAGGETT began practice at the new Lennox Branch of the Donahoe Clinic.

* * * *

DR. DOUGLAS KJELLSEN, formerly of Arlington, is now associated with the Downtown Medical Center, Minneapolis.

* * * *

LECTURERS at the annual two-day seminar sponsored by the Black Hills Medical Society and the University of South Dakota School of Medicine included Drs. Roger A. Harvey, head of the department of radiology, University of Illinois Medical Center, Chicago; Thomas K. Oliver, head of the division of neonatal biology and professor of pediatrics at the University of Washington, Seattle; and Thomas H. Holmes, winner of the American Psychiatric Association Hofheimer Prize and also of the University of Washington, where he is professor of psychiatry.

* * * *

DR. DAVID DIXON MELLENCAMP, a 1966 graduate of the University of Wisconsin Medical School, was assigned to the Rapid City U. S. Public Health Service Indian Hospital (Sioux Sanatorium) as a medical officer.

* * * *

DR. EDWARD C. HANISCH, JR., is on leave of absence from the Huron Clinic staff to study anesthesiology in relation to obstetrics and gynecology at the University of Kansas Medical Center.

* * * *

DR. TACETTIN TUREDI, a native of Turkey, joined the staff of the Herreid City Hospital, Mobridge. He is a heart specialist.

Deaths . . .

DR. AMES W. NASLUND, 66, Edina, Minn., died Aug. 13. A former chief of radiology at St. Barnabas Hospital, Minneapolis, he was a graduate of the University of Minnesota Medical School, where he was a faculty member.

* * * *

DR. OTTO C. MAERCKLEIN, 92, Mott, N.D., died July 25. He had practiced at Adams, Minn., Ashley, and Dickinson, before moving to Mott in 1917. He was an 1896 graduate of the Milwaukee School of Medicine.

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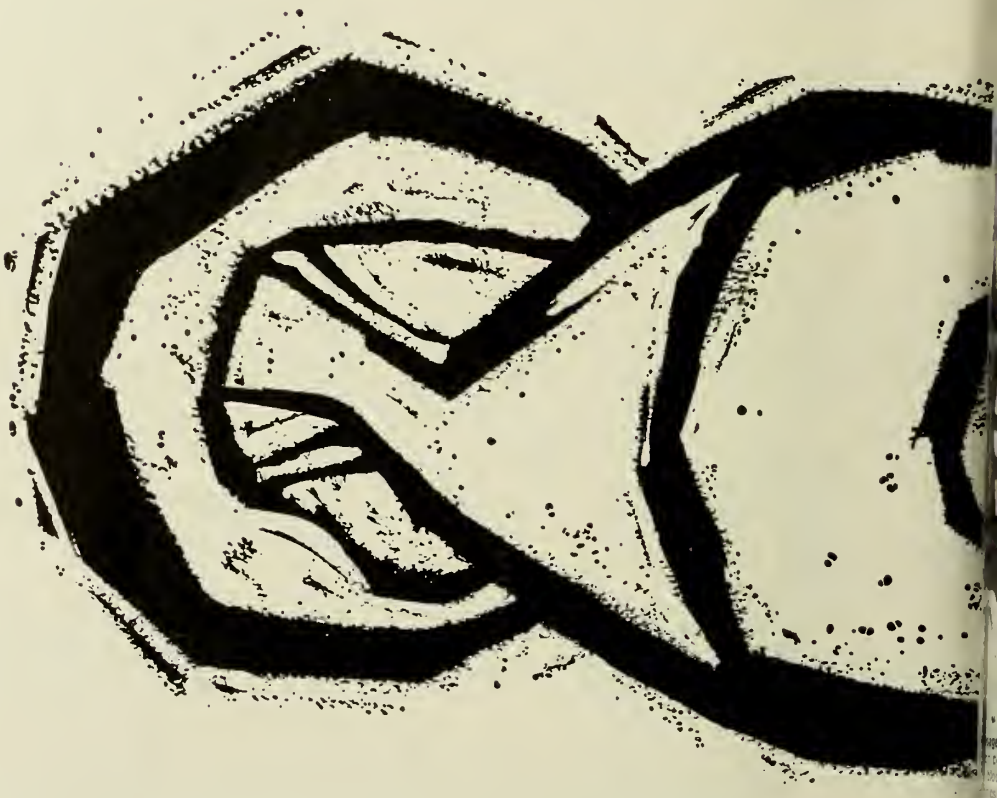
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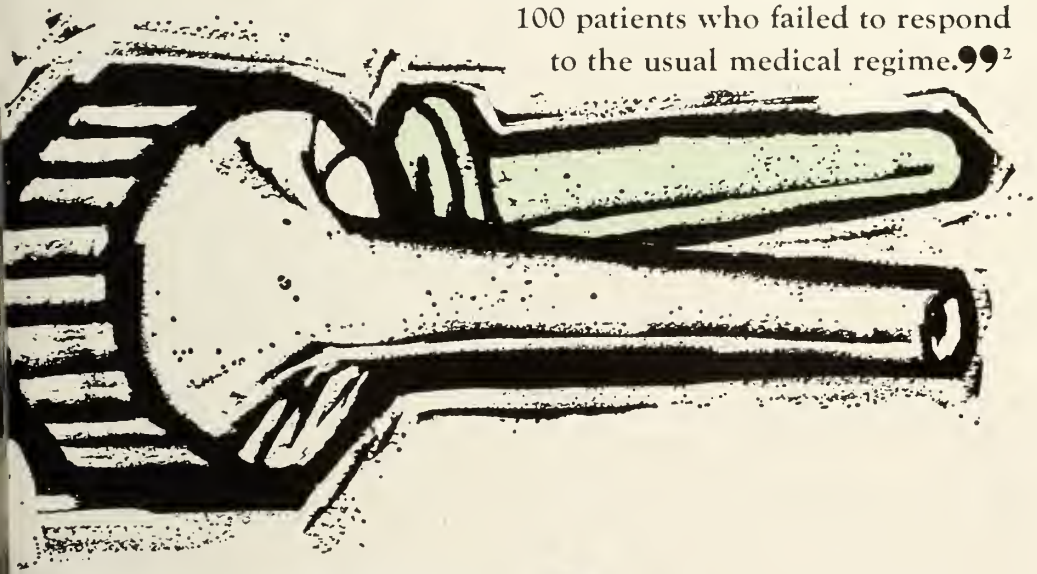
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1. Sherman, L. F.: The treatment of ulcerative colitis with hydrocortisone alcohol retention enemas. A topical and systemic anti-inflammatory agent, *Minn. Med.* 48:605, May 1965.

2. Sherman, L. F., Tenner, R. J. and Schottler, J. L.: Treatment of ulcerative colitis with hydrocortisone alcohol retention enemas: A topical and systemic anti-inflammatory agent. Part II, *Dis. Colon & Rectum* 10:43, January-February 1967.



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References: 1. Musser, Ruth D., and Shubkagel, Betty L.: Pharmacology and Therapeutics, ed. 3, New York, Macmillan Company, 1965, p. 363. **2.** Council on Drugs, American Medical Association: New Drugs 1965, Chicago, American Medical Association, 1965, p. 157. **3.** Modell, Walter (Ed.): Drugs in Current Use 1966, New York, Springer Publishing Company, Inc., 1966, p. 77.

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(1) Bilbao, M. K.; Frische, L. H.; Rösch, J., and Dotter, C. T.: Hypotonic Duodenography, Scientific Exhibit, Radiological Society of North America, Chicago, Nov. 27–Dec. 2, 1966.

(2) Bilbao, M. K.; Frische, L. H.; Dotter, C. T., and Rösch, J.: Hypotonic Duodenography, Radiology, in press.

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CONTENTS

Psychologic Aspects of Amputation of a Lower Limb 429
Elaine B. Simpson, M.A., and Henry F. Albronda, M.D.

Series on Urology for the General Practitioner: Diagnosis of Renal Cyst Versus Renal Tumor 432
George T. Mellinger, M.D.

Bacteremia: Common Pathogens and Methods of Management 439
William J. Martin, M.D.

Oesophagopleural Fistula: A Complication of Lobectomy, Report of an Unusual Case 450
Nit Konsuwan, M.D., and Kasarn Chatikavanich, M.D.

Hyperglycemic Nonketotic Coma Induced by Peritoneal Dialysis 453
Robert Whang, M.D.

Topical Pulmonary Eosinophilia: A Clinical Study 457
R. K. Narang, M.D.

News Briefs 10A

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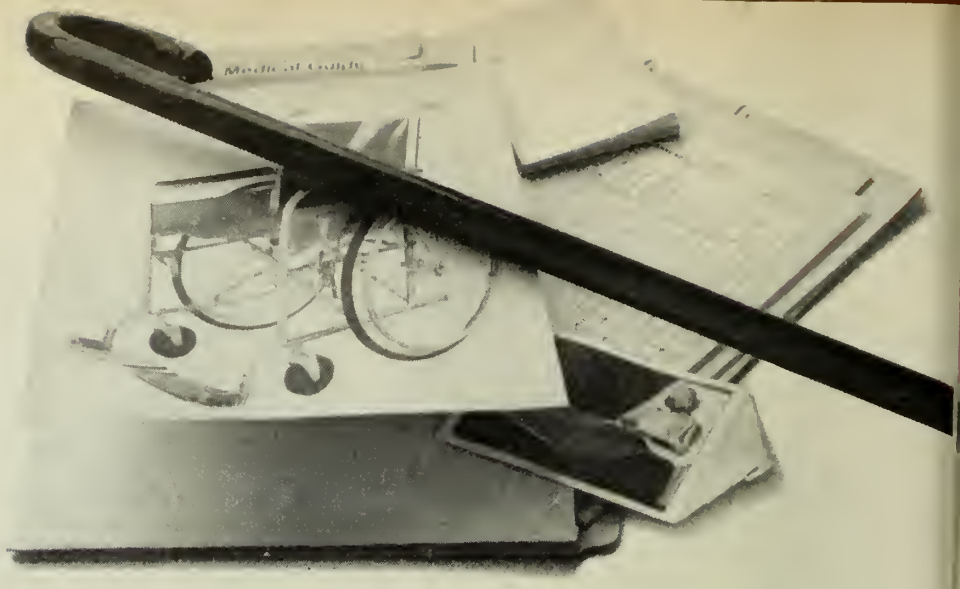
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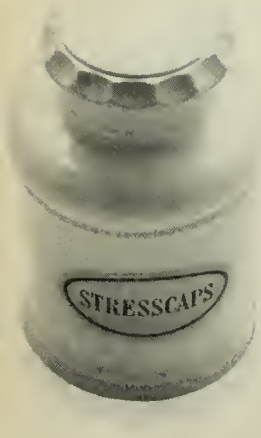
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The Journal-Lancet

SERVES THE MEDICAL PROFESSION OF THE UPPER MIDWEST

Psychologic Aspects of Amputation of a Lower Limb

ELAINE B. SIMPSON, M.A., and
HENRY F. ALBRONDA, M.D.

San Francisco, California

THE CIRCUMSTANCES involved in the experience of losing a limb are many, and amputations are performed for many different reasons. Each of the situations thus arising provides a different emotional effect. A few of the kinds of factors found to be relevant for psychologic adjustment to amputation are as follows: (1) the extent of forewarning given to the person before the actual removal of the limb; (2) the presence of emotional support during critical periods; (3) the quality of the relationship with the surgeon performing the amputation; (4) the general physical condition of the person; (5) his ability to meet the expenses of the amputation; (6) the extent to which occupational and vocational plans were disrupted; (7) the development of fitting problems; (8) the presence of special compensating factors, such as escape from an intolerable battle situation or gain in the form of a generous monetary settlement; and (9) the basic personality trends of the patient which tend to influence his interpretation of what has happened to him and to maximize or to minimize the loss.

ELAINE B. SIMPSON *is clinical psychologist, Berkeley, Calif.* HENRY F. ALBRONDA *(deceased) was assistant professor of psychiatry, University of California School of Medicine; psychiatrist-in-charge, Adult Psychiatry Clinic, University of California Medical Center, San Francisco.*

This paper is based on material collected by the psychology study group of the Biomechanics Laboratory of the University of California. The information was obtained by means of questionnaires, interviews by psychiatrists and psychologists, and by psychologic testing.

All the subjects were paid volunteers and were ambulatory, but otherwise were a heterogeneous group composed of veterans and nonveterans, men and women 19 to 84 years of age, persons who had lost a leg within the year, and persons who had had that experience as long as forty years ago. They were people with a wide variety of medical, social, and emotional experiences.

In the interviews each person was asked to give some general background and identifying information about himself and then to tell in detail what led to the amputation, including the medical facts as the subject understood them, and the subsequent developments with regard to the stump, fitting for prosthesis, return to work, social adjustment, and so on. If, in the course of giving this history, the subject made no spontaneous mention of feelings about the various events, he was asked to tell about his emotional reactions. Encouragement and specific questions were used if necessary. The persistence with which the examiner sought psychologic information varied. The subject's ease in speaking

of emotions was the main factor guiding the examiner, who hoped to avoid the superficial or defensive answers of persons under pressure to reveal more about themselves than they care to. Some persons, although open and cooperative, seemed entirely unused to introspection and appeared to be startled by questions about their psychologic reactions, as if in the midst of the stress and concern of the time of amputation they had never considered how they felt about it. At the other extreme were a few subjects who had spent much time thinking about psychologic problems of amputation. Some were highly intelligent, sophisticated, articulate people who, in recounting their own experiences, did much to amplify and clarify the brief statements of less articulate subjects.

EARLY REACTIONS

What were the subject's very earliest reactions to the idea of amputation or to finding the leg actually gone? Quoting the subjects themselves we find them recalling a wide range of reactions.

Disbelief. "It didn't enter my mind they'd take my leg off—I guess I wouldn't let it."

"The doctor had mentioned in cases like mine, amputation sometimes became necessary but it never occurred to me at the time to apply that to myself, that I'd lose a leg."

"I was pretty doped up at the time and I thought, 'Oh yeah, the doctor's telling you foolish things.'"

Stunned feelings. A boy of 14 years at the time of amputation recalls, "It was sort of a shock to have the leg gone."

He felt that his leg was still intact because of the phantom-limb sensation; then he would look down and see a stump on the pillow and be shocked.

Fear and panic. "I'd wake up from nightmares in a cold sweat and not remember what scared me."

Another patient, after a railroad accident, awakened in the hospital on his way to the operating room screaming and pleading with attendants not to take his legs off, not to amputate.

Rage and anger. "I discovered the leg was gone and I got mad and was cussing people out and giving the doctor hell. We got bombed again that night and I was hoping they'd drop one of the damn things right in the middle of the tent."

Another subject said that while he was still very weak and nearly immobilized after the operation, he demanded to know whether the leg had been removed, and when told that such had been done, sat bolt upright. He had no memory for the rest of the experience, but was told that he started to call his wife names and to curse the nurses and physicians who came to help.

Another subject was "... mostly mad at the damn injustice . . . nothing to vent my spite on, made it harder."

Grief and despair. "I intended as soon as I could get in my car again to drive to the bridge."

"I lost all desire to live for a while."

"I still have a lump in my throat all the time. I suppose time will help me accept it."

Another subject said he told the surgeon beforehand, "If you are going to amputate my leg I'll end my life." He felt like "a big animal caught in a cage."

Relief and happiness. "It was a relief to get rid of that pain."

"I was very happy. I thanked the doctor." (This was after months of suffering while attempts were being made to save a damaged leg.)

Another subject, who also had suffered much before the amputation, said, "I felt very peaceful, somehow relieved of something, a burden, something I couldn't stand any more."

Horror or revulsion. For instance, the young man who saw his stump for the first time and thought it looked like "a big old steak" and cried, must have been filled with revulsion as well as with grief or, as he himself referred to it, with "self-pity."

Shame or guilt. One man speculated that since he had mistreated animals on his father's farm as a child, he might be suffering punishment for those cruelties—since he could not think of any other wrong he had committed.

ADJUSTMENT TO AMPUTATION

Aside from these early reactions, how did the subjects cope with the fact of amputation? Control of strong feelings and acceptance of the reality of the new problems were seldom accomplished immediately and rationally. One patient said that it was a week or so after the amputation before he actually realized that the leg was gone and gone forever. Others postponed asking about an extremity or looking to see for themselves if the leg was gone after operation, delaying consideration of that fact for hours or, in some cases, for days.

One woman said she did not react until the fourteenth day when the stitches were removed. Then she cried for an hour and ended by shrugging her shoulders and saying between sobs, "I guess I just have to get used to it like false teeth."

In other cases, also, a sudden discharge of

feelings marked a turning point in the course of psychologic recovery.

The tendency to postpone or to deny the significance of the loss may influence rehabilitation. One patient who recently underwent removal of both legs repeated many times in an almost pleading voice, "I have everything I had before. The lack of legs doesn't bother me. A man is a man, not legs."

Another, whose leg had been removed ten years previously, said, "Friends I'd hear talking behind my back that I'd never be the same again. I proved to them—not just to prove it to them—I did everything I had done, rode a motorcycle; I'm the same as ten years ago."

This kind of denial actually may help rehabilitation or it may lead to dangerous nonadaptive behavior such as, for example, that of one subject who said in an almost boastful tone that he had managed to walk even when there was a blister the size of his hand on his stump and the stump itself was black and blue. He was active in his recreation and stacked heavy packages at work, standing for four to six hours at a time. He sustained most of his weight on the remaining leg and said he was "working the heck out of it." He defended his behavior by saying, "It's either that or sitting down and getting nervous."

Psychologic aftereffects were reported by some of the patients. Some said they had gone through periods of depression, overactivity, bitterness, or alcoholism in reaction to the amputation or to subsequent difficulties in readjustment. Others referred to being "all shook up," unable to cope with problems, or "not themselves" in some other respect for varying periods after the amputation. "Nervousness" sometimes accompanied physical illness, and it is open to speculation to what extent emotional turmoil lowered resistance to disease and was a factor in illnesses reported during the convalescent period.

COMMENT

Like any other distressing event, amputation produces an effect on a person's state of mind and adjustment which will be determined in part by the sum of his previous experience as it is organized and expressed in his personality and in part by the nature of the experiences he meets. The person who has little sense of his own worth, has had repeated disappointments and deprivations, and has relatively few sources of satisfaction in his present life will find it proportionately more difficult to accept a new loss and to make the necessary readjustments. Clinical intuition will not tell us the relative importance of each factor in respect to patients deprived of an extremity in general, nor will it guarantee that we shall think of all the relevant factors. It is hoped that research conducted with statistical and experimental approaches will help in this regard, focusing our observations and sharpening our ability to predict individual reactions so that we shall be able to anticipate which persons will need special help in the process of psychologic recovery.

Read before the Section on Physical Medicine and Rehabilitation, American Medical Association, June 23, 1964, San Francisco, Calif., as part of a symposium on management of lower-extremity amputees.

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Diagnosis of Renal Cyst Versus Renal Tumor

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FOR MANY YEARS the operating table was considered the proper place to resolve the differential diagnosis of cyst versus tumor of the kidney. Recently, due to refinements of X-ray techniques and the introduction of newer and better instruments, advanced radiological procedures have been purported to aid in making the proper diagnosis. As performed at this hospital, translumbar aortography left something to be desired, but the newer transfemoral catheterization techniques are quite satisfactory. Renal planigraphy and tomography are helpful, but this author has not been willing to rely completely upon them. Renal cystography is convincing, particularly with contrast films made by intracystic injection of CO_2. On the other hand, insertion of a needle into a tumor may be harmful.

Radiological studies fail to answer certain questions. Therefore, beginning a few years ago at this hospital, whenever a renal mass or filling defect was found, this author made it a policy to perform a diagnostic work-up such as was considered proper and necessary at that time to afford a nonoperative diagnosis of renal cyst or renal neoplasm. The films and history of each patient were then reviewed by the radiology service, the author, and 2 or more urological consultants. The diagnosis made by each was considered a preoperative diagnosis. Then all patients who were in condition to withstand nephrectomy were operated upon and the involved kidney was exposed. Only then was the final diagnosis made. This policy has been in effect for a little over five years and during this period a total of 85 patients with renal masses were operated upon (Tables 1-3). We now present 12 interesting cases.

CASE REPORTS

Case 1. This 43-year-old male was referred to us for gallbladder exploration, with a provisional diagnosis of pancreatitis with or without choledocholithiasis. A compression fracture of D-7 was found and the history of a fall two months earlier was obtained. Gastrointestinal series, barium enema, and bone marrow examination

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TABLE 1

RENAL MASSES EXPLORED
Jan. 1, 1961 to Jan. 1, 1966

	Diagnosis	
	Pre-operative	*Post-operative*
Renal cyst	25	36
Renal mass (type undetermined)	17	0
Hypernephroma	42	39
Renal abscess	1	2
Granuloma of kidney	0	1
Renal infarct	0	1
Renal adenoma	0	1
Pyelonephritis	0	1
Papillary tumor of renal pelvis	0	1
Fetal lobulation of kidney	0	1
Normal kidney	1	2
Total renal masses explored	85	

TABLE 2

OPERATIONS PERFORMED ON RENAL MASSES

Nephrectomy	45
Resection of renal cyst	34
Renal biopsy	3
I and D of renal abscess	2
Exploration	1
Total masses operated upon	85

TABLE 3

CAUSES OF DEATH WITHIN 2 MONTHS OF OPERATION

Myocardial infarct PO XV	1
Pneumonia (atelectasis?) PO I	1
Wound infection PO V	1
Total deaths	3

(all patients who died had hypernephroma)
Death rate—3.5% in 2 months



Fig. 1. Case 1. Retrograde pyelogram

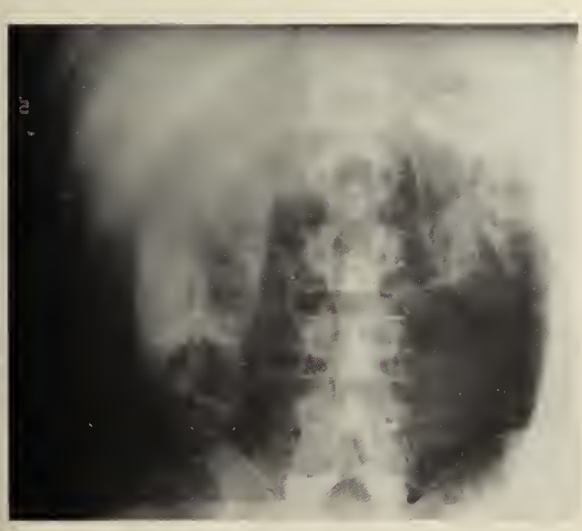


Fig. 2. Case 1. Aortogram

were normal. Intravenous urogram showed a filling defect at the upper pole of the right kidney; retrograde pyelogram (Fig. 1) also showed the mass. Aortography (Fig. 2) showed the avascular mass again. Transabdominal exploration showed a large hypernephroma extending into the right adrenal gland. Postoperative injection of the renal arteries showed the tumor to be avascular. Only the history of the vertebral fracture gave a definite clue to the true diagnosis.

Case 2. This 35-year-old male had a left varicocele for the previous seven years. He was hospitalized following gastrointestinal hemorrhage. A psychiatrist diagnosed a schizoid-type personality.

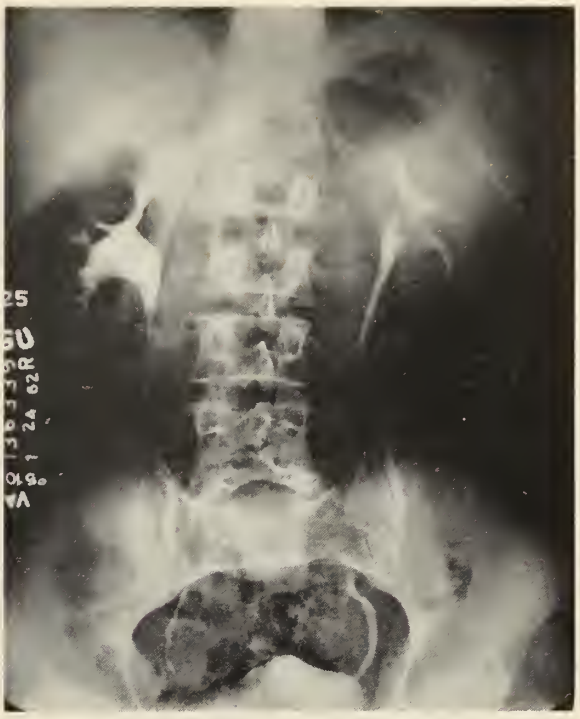


Fig. 3. Case 2. Intravenous urogram

Work-up for the gastrointestinal bleeding (Fig. 3) showed only a mass at the upper pole of the right kidney. Retrograde pyelogram demonstrated a similar mass. Translumbar aortogram was of poor quality. Surgical exploration showed, at the upper pole of the right kidney, a large blue mass which appeared to be a cyst filled with old, clotted blood. This was diagnosed by the surgeon as hemorrhagic renal cyst and the kidney was removed. In the pathologist's words, "The cyst fluid was gray-green. The cyst cavity was filled with clotted blood. Diagnosis: Benign cyst of the kidney." The cause of the hemorrhage was unknown.

Case 3. This 71-year-old male was admitted to the hospital because of total gross painless hematuria. Intravenous urogram demonstrated a mass in the region of the left kidney with no function noted on that side. Retrograde pyelogram (Fig. 4) also showed the mass. Aortogram showed no tumor staining and poor vascularity of this area. Exploration revealed a large hypernephroma.

Case 4. This 45-year-old male had chronic low backache. Intravenous urography and retrograde pyelography, performed before referral to us, showed a mass at the lower pole of the left kidney, producing only mild calyceal deformity. Translumbar aortogram (Fig. 5) showed an avascular mass. Exploration disclosed a cyst which was 10 cm. in diameter and filled with dark fluid and blood clots. Nephrectomy was performed and the histological diagnosis was hypernephroma. Possibly this was cystic degeneration of tumor.

Case 5. This 68-year-old male had a prostatic resection. Routine intravenous urogram (Fig. 6) showed stretching of the calyces of the midportion of the right kidney. This appearance was confirmed by retrograde pyelograms. A translumbar aortogram suggested "a peculiar vascular mottling" in this same area. Retro-



Fig. 4. Case 3. Retrograde pyelogram

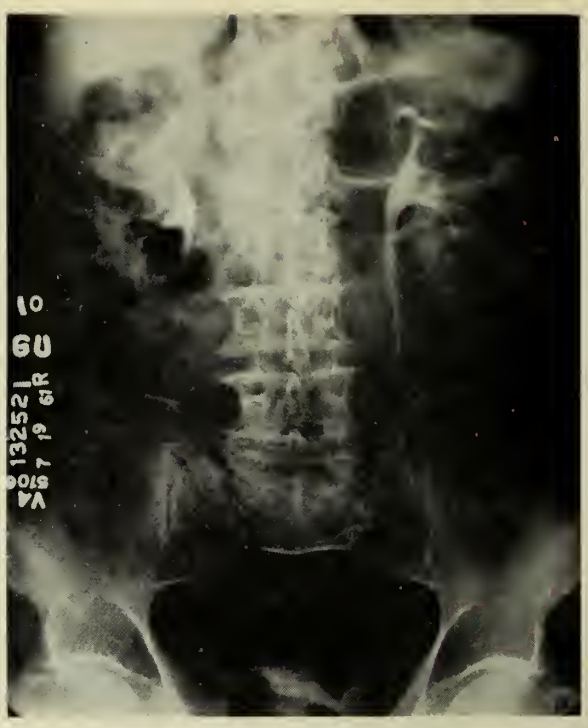


Fig. 6. Case 5. Intravenous urogram

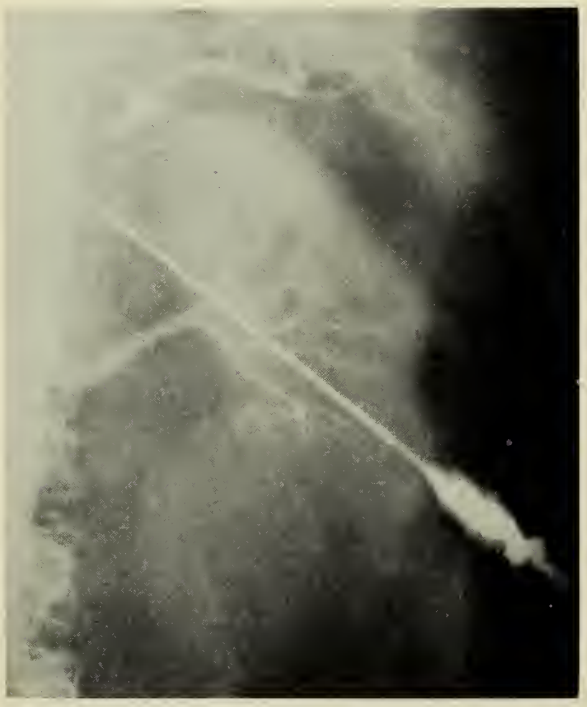


Fig. 5. Case 4. Translumbar aortogram



Fig. 7. Case 6. Retrograde pyelogram

grade aortography again showed suspicious areas. However, the renal outline appeared normal. At exploration the kidney looked normal but a suspicious hardness and mass were felt in the midportion of the kidney. The pathologist's diagnosis was: "Normal kidney." We cannot account for the "tumor staining."

Case 6. This 41-year-old male school bus driver had intermittent, total, gross, painless hematuria. Intravenous urogram showed a space-occupying lesion at the lower and middle portions of the right kidney. Retrograde pyelogram (Fig. 7) confirmed this. Translumbar aortogram indicated an avascularity of the same area, and retrograde aortogram, of better quality, supported this. Surgical exploration showed a mass. Nephrectomy was



Fig. 8. Case 7. Intravenous urogram



Fig. 9. Case 8. Percutaneous aortogram

performed. Pathologist's diagnosis: "Hypernephroma."

Case 7. This 70-year-old male had gross hematuria. A transitional-cell carcinoma of the bladder was excised transurethrally. An intravenous urogram (Fig. 8) demonstrated spreading of the calyces of the lower pole of the left kidney. Chest X-ray was read as "an ill-defined nodular density overlying the anterior end of the right third rib which was not present on previous examination." Aortography was described as showing stretching of several arterial branches. Our diagnosis: Cyst. Radiologist's diagnosis: Cyst with likelihood of small tumor. Exploration showed fetal lobulation; no cyst or tumor was present.

Case 8. This 72-year-old male was admitted to the hospital because of gross painless hematuria. KUB showed a large space-occupying lesion at the lower pole of the right kidney. Retrograde pyelogram pictured the kidney pushed upward and laterally. Aortography (Fig. 9) showed an avascular lesion and delayed film made this appear definitely as a cyst. This cyst was punctured and a renal cystogram (Fig. 10) was performed. Exploration disclosed a large cyst with much old blood, and nephrectomy was performed. The specimen showed a large cyst containing hypernephroma. Only the cystograms hinted at the true diagnosis.

Case 9. This 50-year-old male had a history of gross painless hematuria. He was quite obese but physical examination was essentially normal. Intravenous pyelogram showed a mass in the midportion of the right kidney, confirmed by retrograde pyelography (Fig. 11). Aortography demonstrated avascularity in the region of this mass (Fig. 12). Late films definitely showed the appearance described as being due to cyst. At surgical exploration there was a bluish-brown mass which appeared to be a hemorrhagic cyst and nephrectomy was performed. Pathologist's final diagnosis: "Transitional-cell carcinoma of the renal pelvis." Ureterectomy was performed subsequently.

Case 10. This 66-year-old male was admitted to the hospital because of gross hematuria for five days. Intravenous urogram (Fig. 13) showed spreading of the calyces with a calcified area above the right kidney. Retrograde pyelogram confirmed this. A cholecystogram pictured the opacity outside of the biliary tract. Planigram suggested a mass in the lower and midportion of the kidney. Aortography showed spreading of the vessels around an avascular lesion. Delayed films substantiated the appearance of cyst. At renal exploration a cyst appeared at the lower and middle portions of the kidney; also there was a whitish, depressed, scarred area which seemed to be infarcted. Further exploration proved the calcified area to be outside the kidney but connected to it by fibrosis. Excision biopsy showed hypernephroma and the kidney was removed. The specimen contained that which the pathologist interpreted grossly as infarct, but histological study revealed that the "infarct" was really hypernephroma. Both hypernephroma and cyst were present in the same kidney and were in no way related to one another.

Case 11. This 70-year-old male was seen because of urinary frequency. His prostate was firm but not nodular. Needle biopsies were performed on 2 occasions and only benign prostatic hyperplasia was reported. His blood pressure was 180/74. A KUB roentgenogram revealed a calcification in the region of the left kidney and an intravenous urogram (Fig. 14) showed the calcification overlying the kidney. Retrograde pyelogram added nothing to our knowledge and the left lateral retrograde pyelogram (Fig. 15) indicated the calcified area to be flat and anterior to the kidney.

Nephrotomogram revealed a picture similar to those seen before, while aortogram appeared normal.

The left flank was explored. The kidney looked normal, but there was a calcified pedunculated cyst hanging down in front of the kidney from the anterior superior edge of the left adrenal gland. The cyst was excised. We feel that no measure short of surgical exploration could have given us the correct diagnosis.

Case 12. This 70-year-old male was seen because of prostatism and left pyelonephritis following transurethral excision of a prostatic urethral papilloma. Intravenous urogram appeared to show a lesion in the upper portion of the left kidney. Translumbar aortogram demonstrated tumor staining but a solid lump was seen on the lateral

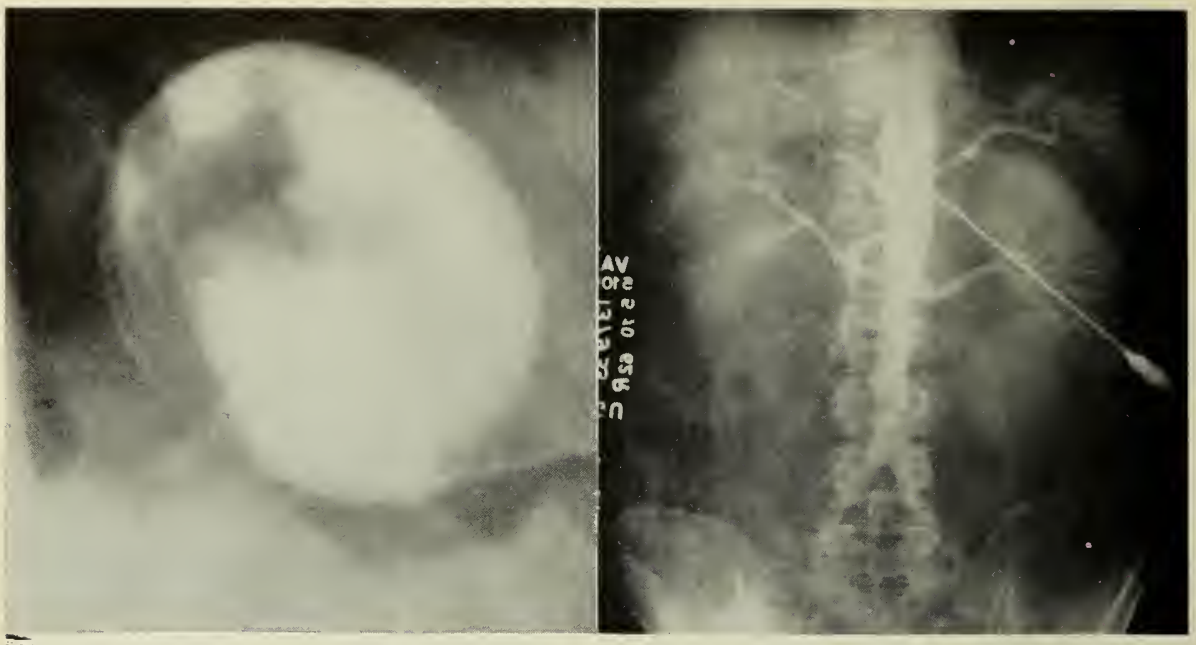


Fig. 10. *(Left)* Case 8. Renal cystogram, patient in lateral decubitus. *(Right)* Case 4. Translumbar aortogram



Fig. 11. Case 9. Retrograde pyelogram



Fig. 12. Case 9. Percutaneous aortogram

portion of the left kidney. Surgical exploration revealed a normal kidney with a small lobe on the anterior surface. Biopsy showed a normal kidney.

We do not argue against the place of roentgenographic procedures alone as preferable for diagnosis of renal pathology in certain poor-risk patients. However, it is our opinion that the preceding cases demonstrate good reasons for the author's preference for open exploration in patients who are considered good surgical risks. We feel that nonoperative diagnosis of renal cyst should be made only in those patients who either will not or cannot withstand surgery because of age or poor general condition. It is our opinion that the risk entailed by surgical exploration of a kidney is less serious than the risk of missing a hypernephroma. Even surgical exploration will not always readily demonstrate



Fig. 13. Case 10. Intravenous urogram



Fig. 14. Case 11. Intravenous urogram

the true pathological picture, as witnessed in Cases 2, 5, 9, and 10, but certainly even in these instances the operative diagnosis was more nearly correct than the nonsurgical diagnosis.

Of 86 patients whose kidneys were explored or removed, only 3 died from any cause whatsoever within two months following the procedure. Almost all of these operations were performed by the resident staff in training at this hospital. In the hands of a urologist who has completed his training, this mortality should be even lower.

We do not argue that any examination, including open exploration, should be performed to the exclusion of all others, and we feel that each method of diagnosis has certain possibilities of error. We wish to discuss these possibilities in greater detail.

Essentially, any roentgenographic diagnostic procedure consists in interpretation of differences in density of the various tissues through which the X-ray beam must pass. Only when there is a difference in radiodensity or opacity will diagnoses be possible. The greater opacity or radiodensity of the kidney, as compared to the surrounding perirenal fat, produces the appearance of the outline of the kidney as seen on KUB films. Planigraphy, or serial "cuts" of the kidney, merely eliminates haziness and distortion from other organs and scatter rays, making this outline more obvious. If a mass of



Fig. 15. Case 11. Left lateral retrograde pyelogram

greater or lesser density from the perirenal fat extends outside of the kidney, its outline should be seen as a "lump" on or near the kidney.

Pyelograms consist of the picture obtained when filling the renal pelvis and calyces, and sometimes the collecting ducts and tubules, with a relatively opaque dye. Such films will show the distortion of the pelvis or calyces produced by masses within the kidney. Masses which produce no pressure on the calyces and those which produce no distortion may be missed.

Nephrography is essentially an early phase of intravenous pyelography, and consists of filling the blood vessels, glomeruli, and tubules with dye, thus making the renal shadow more radiopaque in comparison with the surrounding perirenal fat. Such a film makes detection of irregularities of renal outline much easier.

When the kidney is filled with radiopaque dye (nephrogram) and X-rays are made using a planigraphic technique, the procedure is called nephrotomography. It combines the advantages of both of these film techniques and more accurately shows the density of the intrarenal structure and renal outline. Nephrotomography cannot diagnose tumor or any renal lesion other than the complete absence of the kidney examined. It merely demonstrates differences in density caused by the lesions.

Aortography or, more properly, renal angiography, consists of X-rays made during the filling of the renal arteries with radiopaque dye. These films show the density produced by the filled vessels and, therefore, the outline of the arteries. Renal cysts usually push the renal arteries aside, leaving a relatively avascular area in the kidney. Such vascular defects may be produced not only by cysts but by many other lesions, including infarcts, abscesses, and tumors. Since a good nephrogram is usually obtainable late in the aortogram, the doctor should take advantage of this to determine the relative density of an avascular mass as compared to the surrounding tissue and kidney. Difference in roentgenographic density may be produced by changes in gas shadows in the bowel anterior to the kidney. Such shadows may also be seen, although less well-defined, in tomograms. The actual time of radiologic exposure in these cases is so short that it is not at all inconceivable (and rather probable) that gas shadows would appear in a single area and show no motion.

Only by the performance of puncture of the renal cyst after nephrotomography and aortography may the urologist or radiologist be fairly certain that he has seen a cyst, but it is possible that this diminished density may be due to a tumor of less density, or there may be a tumor within the cyst. We feel that it is virtually impossible to puncture a cyst or tumor without spilling some cells into the surrounding tissue. We do not wish to take even a 1% chance of spreading the tumor, but we also feel that we cannot afford to discharge the patient with only a probable diagnosis. Also, as in our Case No. 10, it is possible that a patient may have both a hypernephroma and a cyst in different parts of the same kidney. If only puncture of the cyst is carried out, the tumor will be left.

Usually, it is only by open surgical exploration that a relatively accurate diagnosis may be made. Suspected lesions may be biopsied or the kidney may be removed. If a perirenal cyst is thought to be present, puncture of the cyst and open exploration may be carried out and cystograms may be obtained before the wound is closed. Even surgical exploration will not always demonstrate the true pathological picture, as witnessed in Cases 2, 5, 9, and 10. But certainly—and even in these cases—surgical diagnosis was more nearly correct than the preoperative radiological diagnosis.

We wish to emphasize that complete and total accuracy in the diagnosis of renal masses is not possible; that each method, whether radiological or surgical, has its limitation and yields only a measure of accuracy. Other factors being equal, the presence of gross hematuria would make us lean more toward a diagnosis of renal malignancy than would the absence of such a history. It is our opinion that, all things considered, the open surgical exploration of renal masses provides the most accuracy. However, in poor-risk patients the relative inaccuracy of radiological diagnosis, as compared to open surgical evaluation, may have to be accepted, but this relative accuracy must be realized, considered, and not overvalued.

Bacteremia: Common Pathogens and Methods of Management

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Although almost any bacterial species can produce bacteremia under certain circumstances, some species are more frequently responsible. The present paper will consider only the more common species. Infections in which antibacterial therapy is unavailing are not considered here. One should discard from this group of infections those instances of transient bacteremia, in which bacterial invasion of the blood is fleeting and clinically insignificant, as may follow dental or urologic manipulation. Also, one should distinguish between bacteremia per se and those situations in which it is a manifestation of endocarditis.

DIAGNOSTIC AND THERAPEUTIC CONSIDERATIONS

Knowledge of the location of a primary nidus of infection, of the existence of an underlying, predisposing disease, and of any prior therapy may suggest the type of pathogen likely to be responsible for a given case of bacteremia. Such information may allow initiation of more reasonable antibacterial therapy until definitive microbiologic data are forthcoming and also may be important in determining other aspects of management, such as surgical drainage.

The antibacterial regimen selected for use in a given instance depends on many factors, such as the severity of the infection, the patient's general condition and resistance, his allergic diathesis, and his ability to accept the regimen by the route designated. In general, combinations of antibacterial agents should be reserved for situations that demand treatment while microbiologic data are awaited, for polymicrobic infections, and for instances of bacteremia wherein there are indications that an enhanced effect is possible.

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PRINCIPLES OF MANAGEMENT

The principles of management of patients having bacteremia can be summarized as follows: [1] procurement of samples of blood for adequate microbiologic study and initiation of treatment empirically if the condition of the patient warrants, [2] reevaluation if in vitro bacterial susceptibility tests later indicate poor choice of an antimicrobial regimen but continuation of the regimen unaltered if the patient is doing well, [3] in severe infections, procurement of serum bactericidal tests in a further attempt to determine if the bacteria are eradicable by the antimicrobial regimen employed, [4] administration of the drug or drugs in adequate dosage for a sufficient time, and [5] correction of any condition predisposing to, or complicating, the infection.

Staphylococcal bacteremia. At the outset in a discussion of staphylococcal bacteremia it may be well to reiterate that the belief which equates coagulase-positivity with staphylococcal pathogenicity and relegates coagulase-negative staphylococci to the position of harmless saprophytes is fallacious. However, coagulase-negative staphylococci are more apt to cause bacterial endocarditis, especially in postcardiotomy patients, or meningitis in patients who recently have had ventricular shunt procedures than to cause bacteremia. When coagulase-negative staphylococci are causative of bacteremia, the victims usually are those with decreased host resistance, such as may characterize certain hematologic diseases.

In a series of patients with bacteremia due to Staphylococcus aureus studied by members of our group[1] it was found that more than 90% had had either a preceding operation or a recognized underlying illness.

Of the nonsurgical patients in this group, which amounted to almost one-half of the total studied, about 25% had carbuncles, furuncles, or

cellulitis; approximately 20% had malignant lesions; 10% had "collagen diseases"; and 10% had generalized cutaneous disease. About 60% of the patients in whom invasion of the bloodstream occurred after operation had undergone surgical procedures on the genitourinary tract. About 20% of the remaining surgical patients had undergone orthopedic or neurologic procedures, and about 10% had had operations on the gastrointestinal tract.

Conditions of a suppurative nature, such as epidural abscess, septic arthritis, and purulent pericarditis, occurred in about 10% of patients and were classified as metastatic complications of the bacteremia. Hypotension or bacteremic shock was noted in about 5% of patients.

Although there is no antibiotic that is a panacea for staphylococcal bacteremia, knowledgeable manipulation of the antibiotics[2] to which these organisms are sensitive allows a reasonable chance of obtaining satisfactory results. Penicillin G remains the most effective agent in the treatment of staphylococcal bacteremia, provided the organisms are sensitive to it.

Definitive treatment of staphylococcal bacteremia should be based on the results of in vitro tests for bacterial susceptibility. The need for agents with the potential of the penicillinase-resistant penicillins, such as methicillin, oxacillin, cloxacillin, and nafcillin, is not as great in staphylococcal bacteremia as in staphylococcal endocarditis. It is generally accepted that eradication of staphylococcal endocarditis requires use of agents capable of bactericidal effect, but this requirement does not appear mandatory for eradication of staphylococcal bacteremia. This is so because bacteriostatic agents, such as erythromycin and novobiocin, have been effective in staphylococcal bacteremia.

There is no gross objection to the use of methicillin or an allied drug when cultures indicate staphylococcus to be causative of bacteremia and before the results of susceptibility tests have been reported; when these results become available, one can reevaluate the regimen accordingly.

If the staphylococcus resists the action of penicillin G, that is, if it requires more than 1.0 μ per milliliter for inhibition but is sensitive to erythromycin this drug may be the agent of choice since it is virtually nontoxic. Next, one may wish to consider lincomycin or novobiocin. In the highly improbable situation that erythromycin, lincomycin, or novobiocin cannot be used, then methicillin or an allied drug may be the drug of choice. When these drugs are not applicable, as in patients who have known allergy to penicillin, cephalothin may be tried. Thereafter, kanamycin precedes consideration of vancomycin because, although both are ototoxic, kanamycin is given intramuscularly whereas vancomycin is given intravenously. Even though chloramphenicol can be absorbed when given orally, it follows vancomycin in choice because of its propensity to depress the bone marrow; however, it precedes consideration of ristocetin which also has a hematotoxic effect but has the added disadvantage of requiring intravenous administration. Before or after consideration of chloramphenicol and ristocetin—the decision may depend on one's fear of ill effects on the marrow or it may depend on the laboratory facilities that are available—one can consider combined antibiotic therapy. If such therapy is chosen, one of the so-called bacteriostatic agents such as erythromycin or novobiocin is used, together with a bactericidal agent such as bacitracin or kanamycin. However, the need for such cumbersome therapy is less common with the present availability of several effective antistaphylococcal agents.

Because of the effect of the tetracyclines on the body flora, with possible superinfection due to resistant strains, these drugs should not be given to patients who have staphylococcal bacteremia. Streptomycin, to which staphylococci develop resistance rapidly, and polymyxin B and colistin, which lack significant antistaphylococcal action, usually are not considered in the treatment of staphylococcal bacteremia.

Indiscriminate use of methicillin and allied drugs must be avoided. Since the degree of resistance to an agent is correlated with the amount of the drug being used in a geographic region, and since resistance to these agents has been demonstrated both in vitro and in vivo, the incidence of resistant strains probably will increase. Also, these agents should not be used in patients who have had allergic reactions to other penicillins, since cross-allergenicity is known; nor should they be used in patients with allergic diseases, particularly asthma, unless the need is overriding.

Streptococcal bacteremia. In a series of cases of streptococcal bacteremia without endocarditis encountered during a fifteen-year period by members of our group,[3] group A beta-hemolytic streptococci or Streptococcus pyogenes was the offending organism in about 50%. Group D nonhemolytic streptococci or enterococci, such as Streptococcus faecalis, were responsible in 30% of cases, while alpha-hemolytic streptococci of the viridans type, such as Streptococcus mitis and Streptococcus salivarius, were causative in

the remainder. In almost half of the enterococcal infections, other organisms were found along with the S. faecalis, including S. aureus, Escherichia coli, and Proteus vulgaris.

In half of the patients with bacteremia due to S. pyogenes, there was evidence of preceding suppurative conditions elsewhere in the body, such as otitis media with mastoiditis, streptococcal pharyngitis, and erysipelas. Complications of streptococcal bacteremia were noted only in those patients whose infection was due to S. pyogenes, and these included suppurative polyarthritis, suppurative ophthalmitis, mycotic aneurysm, and acute hemorrhagic nephritis.

Of the patients whose bacteremia was due to an alpha-hemolytic streptococcus such as S. mitis, surgical procedures, diabetes mellitus, peritonitis, and preceding sore throats appeared to be etiologically significant.

The patients whose bacteremia was due to enterococci stand in sharp contrast to the remaining patients of the streptococcal series in that the apparent portals of entry were solely gastrointestinal or genitourinary. Of the patients whose infection may have arisen via the genitourinary tract, the majority had undergone transurethral resection or manipulation. Of the patients in whom the gastrointestinal tract appeared to be the likely port of entry, acute gastroenteritis, biliary stricture, gastric resection, carcinoma of the colon, and dental extraction were included.

In 1938, in a review of cases of bacteremia seen at the Mayo Clinic during a three-year period, the bacteremia in 61 of 144 cases was found to be due to S. pyogenes. In the more recent review, only 34 patients were noted with bacteremia due to all types of streptococci during a fifteen-year period. Therefore, the incidence of streptococcal bacteremia encountered at this clinic apparently decreased from more than 20 cases annually to slightly more than 2 with the advent of modern antibacterial therapy. The decreased incidence of streptococcal bacteremia may be related to the action of antibacterial agents in eliminating the infectious precipitants of that condition.

In a study by members of our group, it was found that bacterial endocarditis due to S. mitis and kindred organisms at the Mayo Clinic had an occurrence rate of about one case per month.[4] Enterococcal endocarditis was found to have an occurrence rate of about one case every four months.[5] It is obvious, then, that streptococcal endocarditis is now being encountered considerably more often at the Mayo Clinic than streptococcal bacteremia.

In the treatment of bacteremia due to S. pyogenes, 1.2 million units of procaine penicillin G given intramuscularly twice daily or more often produces satisfactory results. In attempting to combat bacteremia due to S. mitis and kindred organisms, we have used 1 gm. of streptomycin and 1 million units of procaine penicillin G given intramuscularly every twelve hours, with satisfactory results.[4] However, because of the potential auditory toxicity of streptomycin, members of our group currently prefer to use only 1 gm. of the drug per day but administer 20 million units of aqueous crystalline penicillin G intravenously per day in the management of bacteremia due to alpha-hemolytic streptococci. The general condition of the patient including his renal function, his response to a given regimen, results of serum bactericidal tests, and similar factors may influence the administration and dose of streptomycin in these cases.

Because most strains of enterococci such as S. faecalis are more resistant to penicillin G than are the other strains of streptococci, usually we use at least 20 million units of aqueous crystalline penicillin G by the intravenous route, together with as much as 1 gm. of streptomycin daily when attempting to eradicate that organism. There are preliminary indications that ampicillin may be as effective as streptomycin-penicillin G as an anti-enterococcal regimen. An amount of 4 to 6 gm. of ampicillin should be given daily; streptomycin or kanamycin may further enhance the effect of ampicillin.

When a patient requires more than 6 million units of penicillin daily, one usually resorts to continuous intravenous administration. In such cases, one divides the total daily dose of crystalline penicillin G into equal parts and dissolves each half in 1 liter of 5% dextrose in distilled water. To each liter of solution is added 25 mg. of heparin in an attempt to decrease the incidence of chemical phlebitis. The apparatus for administering the solution usually can be adjusted so that the 2 liters are given in twenty-four hours.

If doubt arises as to the merit of the regimen being used in a case of enterococcal bacteremia, the so-called serum bactericidal test may be used. In this test, the patient's serum containing the given antibiotics is assayed against the organism isolated from the patient's bloodstream. If the patient's serum, diluted one to four, does not produce a bactericidal effect on the organism, one increases the dose of penicillin in increments of 10 million units until such an effect is produced.

In streptococcal bacteremia, as in most other

types of bacteremia, treatment is continued until three successive negative blood cultures have been obtained and the patient has remained afebrile for at least seventy-two hours.

If bacteremia due to enterococci is associated with a gram-negative bacillus, antibiotic therapy can be initiated empirically with a tetracycline, chloramphenicol, or cephalothin in addition to penicillin G and streptomycin. If in vitro susceptibility tests later indicate that the empiric choice of drugs is not optimal, the program can be reevaluated.

In patients allergic to penicillin, use of cephalothin, erythromycin, lincomycin, or a tetracycline may be an effective substitute in some cases of streptococcal bacteremia. However, a considerable number of enterococci resist the action of these alternate drugs. Vancomycin and ristocetin may have use as anti-enterococcal agents. Also, about 40% of S. pyogenes resist the action of the tetracyclines and these drugs should not be employed in infections due to these organisms unless in vitro studies indicate their applicability.

Pneumococcal bacteremia. Bacteremia due to Diplococcus pneumoniae[6] may occur during the course of pneumococcal pneumonia, particularly when the infection is fulminating. Endocarditis, most commonly involving the aortic valve, may complicate pneumococcal pneumonia and is characterized by persistent pneumococcal bacteremia. In pneumococcal meningitis at least 50% of patients have bacteremia due to D. pneumoniae. However, invasion of the bloodstream by pneumococci in the absence of pneumonia, endocarditis, or meningitis is unusual. The incidence of pneumococcal bacteremia has greatly decreased since the advent of modern antibacterial therapy. Penicillin G is the drug of choice; the route of administration and daily amount, which may be several million units, are dependent in part on the presence or absence of associated endocarditis or meningitis. In patients allergic to penicillin, drugs such as cephalothin, erythromycin, or lincomycin may be given. Because some strains of pneumococci are resistant to the tetracyclines, use of these drugs in pneumococcal infections without in vitro susceptibility tests indicating their applicability may be hazardous.

Neisserian bacteremia. The clinical management of bacteremia due to Neisseria meningitidis virtually is inseparable from that of meningococcal meningitis. Aqueous crystalline penicillin G in amounts of 1 million units every two to four hours by intramuscular injection may be used but even larger amounts have been given per day intravenously. The possibility of supervening vascular collapse should be borne in mind constantly. Since a number of strains of meningococci are resistant to the sulfonamides, these drugs cannot be relied on for initial management.

Neisseria gonorrhoeae rarely succeeds in invading the bloodstream. Large doses of penicillin G are recommended if invasion occurs.

In patients with Neisserian bacteremia who are allergic to penicillin, erythromycin or a tetracycline is recommended.

Enterobacillary bacteremia. The incidence of bacteremia due to gram-negative bacilli has increased threefold to fivefold in recent years. Among nidi of infection from which bacteremia may originate, in our experience pyelonephritis, cholangitis, and conditions associated with varied degrees of peritonitis are most common. In about half the cases studied by members of our group,[7–9] there was an apparent precipitating surgical or instrumental procedure; in two-thirds of cases, this involved the genitourinary tract and in one-third, the gastrointestinal tract. The underlying noninfectious diseases, which were present in a significant percentage of our cases, included advanced arteriosclerosis, 20%; malignant disease,[10] 10%; hematologic disease,[10] 15%; diabetes mellitus,[11] 10%; and hepatic cirrhosis,[12] 5%. In addition to operation and manipulation, treatment capable of altering the defense mechanisms of the host had been used, including administration of adrenocorticosteroids in 15% of the cases and antimetabolic agents in 5%. Prior effects of certain forms of antimicrobial therapy, such as penicillin-induced suppression of the gram-positive elements of the bacterial flora, appeared to be a factor in the genesis of bacteremia in 20% of the cases. Roentgen therapy had been given to less than 5% of patients in whom bacteremia subsequently was diagnosed. In some cases these possibly predisposing or precipitating factors were multiple. Knowledge of predisposing factors and awareness of the possible supervention of bacteremia due to gram-negative bacilli in susceptible individuals may facilitate early diagnosis and initiation of appropriate treatment.

Members of the enterobacteriaceae continue to be the most common cause of bacteremia due to gram-negative bacilli. These organisms cause about two-thirds of the enterobacillary bacteremias, with E. coli being responsible about twice as often as Klebsiella-Aerobacter organisms. The remaining cases are most commonly due to Proteus organisms. Organisms of this group which are less commonly responsible include Paracolo-

bactrum, Alcaligenes, Salmonella, and Shigella. Certain nonmembers of this group, such as Pseudomonas and Bacteroides are referred to occasionally in this section, especially in respect to their susceptibility to empiric antibiotic therapy.

Our practice has been to initiate antibiotic therapy empirically after clinical examination or on receipt of reports of blood cultures positive for gram-negative bacilli. If in vitro susceptibility tests later indicate the choice of treatment has been less than optimal, one reevaluates the program. A tetracycline combined with streptomycin or, in the more recent past, the conjoint use of chloramphenicol and kanamycin has been the empiric regimen most commonly employed by members of our group under these circumstances. Currently we are investigating colistin or polymyxin B given together with cephalothin and, thus far, the combination has been very satisfactory.

In management of these patients, these antibiotics are given as follows: On receipt of reports of positive blood cultures or, after blood has been obtained for cultures in patients suspected of having bacteremia due to gram-negative bacilli, 500 mg. of a tetracycline are given by mouth every six hours if circulatory collapse appears not so severe as to prevent its intestinal absorption. Chloramphenicol may be given instead, orally in amounts of 0.5 gm. every four hours, if staphylococci also are present, as in patients having furunculosis, or if superinfection with them seems especially likely. Again, chloramphenicol may be substituted if there has been inadequate response after about a twelve- to twenty-four-hour trial of tetracycline or if in vitro data later indicate greater susceptibility to chloramphenicol and there has been poor response to the tetracycline.

Although streptomycin usually is given conjointly with the tetracycline, kanamycin along with chloramphenicol may be substituted when a staphylococcal co-infection is possible or there has been poor response after a twelve- to twenty-four-hour or more trial of a tetracycline and streptomycin. Also, one may consider substituting polymyxin B or colistin when there is underlying hematologic disease or when response is inadequate; in such cases the use of a tetracycline or chloramphenicol usually is continued or either may be replaced by cephalothin. In addition to their effectiveness against Pseudomonas organisms, polymyxin B and colistin have proved efficacious in infections due to other bacilli, such as E. coli and Aerobacter aerogenes. However, some workers avoid the use of polymyxin B and colistin when possible in nonpseudomonal infections because the drugs may produce relatively low serum and tissue levels.

If circulatory collapse appears to be too severe to allow intestinal absorption, a tetracycline is given intravenously in amounts of 500 mg. every eight to twelve hours. Alternatively, chloramphenicol sodium succinate, 0.5 to 1 gm., can be given intravenously every six to twelve hours. As much as 1 gm. of streptomycin—or up to 0.75 gm. of kanamycin if associated staphylococcal infection or refractoriness is noted—is given intramuscularly every twelve hours. When polymyxin B or colistin is used, a maximal dose of 50 mg. is given intramuscularly every six hours. Cephalothin, in amounts of 0.5 to 1 gm., is given intramuscularly or intravenously every four to six hours. If renal function is impaired, less than maximal amounts of the tetracycline, streptomycin, kanamycin, polymyxin B, or colistin are given in an attempt to avoid accumulation in the blood and consequent enhanced toxicity.

In cases of bacteremia likely due to gram-negative bacilli, empiric administration of these regimens, after samples of blood have been obtained for cultures, appears preferable for several reasons. Since identification of bacteria and results of susceptibility tests will not be available for one or more days, the broader antibiotic coverage is desirable, and enhanced antibacterial effect may be obtained.

There is no dispute regarding the generalization that best results are obtained when the infecting organisms are sensitive on in vitro tests to the antimicrobial agents employed, but the difficulty lies in the unavailability of such data when the clinical status of the patient makes initiation of therapy necessary.

With the advent of newer drugs, we have reevaluated our policy of employing combined antibiotic therapy. Colistin, like polymyxin B, has the objection, as a single agent given empirically, that Proteus and Bacteroides organisms usually are resistant. Kanamycin, if used alone empirically, has the therapeutic inadequacy of not being effective against a number of Pseudomonas and most Bacteroides organisms. Ampicillin is inactive against most Proteus organisms that are indole-positive as well as most Klebsiella-Aerobacter and Pseudomonas organisms. Cephalothin is not active against all Klebsiella-Aerobacter, Bacteroides, and indole-positive Proteus organisms and usually is inactive against Pseudomonas organisms. To date, there is no single agent available that assures complete "coverage" against gram-negative bacillary infections.

Disadvantages are possible with the empiric use of these antibacterial regimens. The disadvantages include the dual depressive effect on the gram-negative elements of the flora, making superinfection more likely. However, periodic study of smears and cultures of sputum and stools during antibacterial therapy may reveal changes in the flora that might suggest impending superinfection. Streptomycin, kanamycin, polymyxin B, colistin, and possibly cephalothin may be nephrotoxic; streptomycin, kanamycin, polymyxin B, and colistin may produce a curariform effect; streptomycin and kanamycin are also ototoxic; chloramphenicol is hematotoxic; and polymyxin B and colistin may cause cerebellar ataxia and other neurologic complications. In view of current results on in vitro tests of bacterial susceptibility, which will be further discussed later, streptomycin-tetracycline may not provide antibacterial "coverage" to the degree obtainable with alternative regimens. In addition, combined therapy entails the introduction of parenteral injections or use of an increased number of injections and is more expensive.

It may be pertinent to point out that although regard for the potential ototoxicity of streptomycin should be sufficient to disallow its indiscriminate use, one should have greater concern for the toxic potential of kanamycin. Kanamycin is more likely to cause deafness than streptomycin and, like dihydrostreptomycin, may be responsible for deafness of delayed onset. Streptomycin has the relative advantage of being more likely to cause vestibular damage, which to a considerable degree can be compensated by visual mechanisms.

Occasionally, although empiric therapy is sufficient to prevent death, the response is incomplete; then, microbiologic data may indicate possible use of a single agent.

In other words, when the patient is not doing well clinically, the empiric regimens of a tetracycline plus streptomycin or chloramphenicol and kanamycin are replaced with the following drugs in keeping with the results of the in vitro susceptibility tests. Ampicillin, cephalothin, polymyxin B, or colistin may be applicable in infections due to E. coli; except for ampicillin, the same drugs are considered against Klebsiella-Aerobacter organisms. As a generalization, strains of E. coli are susceptible to streptomycin, the tetracyclines, and cephalothin in about 50% of cases; these bacilli are susceptible to kanamycin, chloramphenicol, and ampicillin in 80% or more of cases. The same generalization is applicable to Klebsiella-Aerobacter organisms except that only about 10% of these bacilli are susceptible to ampicillin.

Penicillin G, ampicillin, cephalothin, and novobiocin are active against the indole-negative P. mirabilis, which causes 80 to 90% of clinical infections due to Proteus organisms. Cephalothin is active in 10 to 20% of infections caused by indole-positive Proteus organisms, that is, P. vulgaris, P. morganii, and P. rettgeri; chloramphenicol also may be effective against these organisms but differences between in vitro and in vivo results with the drug are frequent. Consequently, kanamycin may be the drug of choice against indole-positive Proteus organisms.

It should be noted that cephalothin may not eradicate infections due to certain gram-negative bacilli despite in vitro bacterial susceptibility to the drug; these bacilli include Salmonella and Shigella organisms as well as Haemophilus influenzae.

In regard to salmonellosis, typhoid fever appears to remain well managed with chloramphenicol. Sometimes an initial "loading" dose is given, but the usual maintenance dose is 500 mg. every four hours orally. The maximal daily dose is given for fourteen days; some workers also recommend conjoint use of streptomycin for the first five days. Corticosteroid therapy is given only to patients with marked toxemia and pyrexia who might die if the toxicity is not brought under control promptly; it should not be used as a routine practice. While at this writing chloramphenicol is favored over ampicillin in the control of fever and early symptoms, there are some preliminary indications that use of ampicillin may be followed by fewer relapses. In bacteremia due to other Salmonella organisms, a tetracycline, ampicillin, or kanamycin may be preferable, depending on in vitro susceptibility tests.

Shigellosis, unlike salmonellosis, almost always is a purely intraluminal enteric infection. However, about 50 cases of bacteremia from Shigella have been documented to date. In such patients tetracycline-streptomycin therapy has been suggested, but ampicillin or kanamycin might be preferable.

As a generalization, antibacterial therapy of bacteremia due to these gram-negative bacilli should be continued until the patient has been afebrile for at least seventy-two hours and three successive blood cultures have been negative.

Pseudomonas bacteremia. Among the gram-negative bacilli that are not classified as enterobacteriaceae, there are the Pseudomonas organisms.[13] In recent years there appears to have been an increase in the incidence of bacteremia

due to Pseudomonas organisms. Today, Pseudomonas organisms appear to cause bacteremia about half again as often as Proteus organisms.

Pseudomonas organisms, although normal inhabitants of the intestinal tract of some persons in small numbers, also are generously distributed in nature and are found in water, on skin surfaces, and in the air. Consequently, these organisms have relatively free access to wounds, drugs, and medical instruments. The increased frequency of infections from Pseudomonas may, at least in part, result from the resistance of the organisms to most of the antimicrobial agents which allows them to emerge as the predominating bacilli when more susceptible bacteria have been eradicated. Notable clinical features may arouse the clinician's suspicion that Pseudomonas is the cause of bacteremia in a given case. First are the skin lesions, referred to as "ecthyma gangrenosum," which are commonly seen in the anogenital region and axillae. These lesions begin as macules or vesicles, become pustular, and eventually slough to form necrotic ulcers. About 10% of our patients with bacteremia from Pseudomonas have had such ecthymatous lesions. Another clinical feature worthy of note is marked leukopenia, at times associated with thrombocytopenia, as has occurred in about 5% of our patients. Jaundice occurs occasionally in patients with bacteremia from Pseudomonas. Petechiae are seen occasionally, whereas bluish-green discoloration of the nail beds is rare. Jaundice may be noted in cases due to other gram-negative bacilli but is of increased frequency in Pseudomonas bacteremia.

Polymyxin B and colistin remain the drugs of choice in the management of bacteremia due to organisms of the Pseudomonas group. Our experiences with Pseudomonas bacteremia in patients with such conditions as advanced malignant, hematologic, or renal disease corroborate the experience of others in that no form of current antimicrobial therapy appears to be capable of preventing death in most cases.

Bacteroides bacteremia. Organisms in the family of Bacteroides include B. fragilis, necrobacilli or B. funduliformis, fusospirochetes, and Streptobacillus moniliformis. Members of our group have described some of our experiences with bacteremia due to certain of these organisms.[14] As a generalization, in our experience these organisms cause bacteremia about half as frequently as do Proteus bacilli.

Diarrhea, beginning before antibiotic therapy and terminating with recovery, occurred in one-third of our cases of bacteremia from Bacteroides. Diarrhea may be noted in cases of bacteremia due to other gram-negative bacilli, but classically it suggests causation by members of the Bacteroides family or by Salmonella organisms. Failure to obtain anaerobic cultures of the blood may conceal the fact that bacteremia due to members of the Bacteroides family is present. In two-thirds of our patients with such bacteremia, incubation times ranged from five to twenty days. Since isolation of the organism required such prolonged incubation of anaerobic cultures, one should observe cultures for three or more weeks before considering them sterile. Because of these peculiarities of Bacteroides, in rare cases of strongly suspected gram-negative bacillary infections of the blood, one is reluctant to discontinue empiric antibiotic therapy until approximately two weeks of treatment have been given.

It is our clinical experience that bacteremia due to Salmonella typhosa and Bacteroides or Brucella organisms requires more time for therapeutic control than do other gram-negative bacillary infections of the blood. If therapy of such bacteremia is not continued for approximately two weeks, the incidence of relapse may be significant. Also, an unexpected delay in clinical response to treatment with appropriate antibacterial agents in cases of bacteremia from Bacteroides should suggest the likelihood of a locus of suppuration. As in bacteremia due to S. aureus and to S. pyogenes, metastatic infections are common in bacteremia due to Bacteroides organisms, and surgical drainage may be required in such instances.

Despite the availability of the newer agents, in management of bacteremia due to either B. fragilis or necrobacilli, a tetracycline often is preferred; penicillin G may be active against certain strains of these bacilli. In cases wherein blood cultures are reported as growing these organisms and the patient has been receiving empiric streptomycin-tetracycline therapy, use of the streptomycin is discontinued. In rat-bite fever and Haverhill fever, which are caused by S. moniliformis, and in the rare instance of infection of the blood due to fusospirochetes, penicillin G is recommended. In the form of rat-bite fever known as sodoku, which is caused by various spirallary organisms, such as Spirillum minus, penicillin G is also the drug of choice.

Brucellosis. In patients with bacteremic brucellosis,[15] streptomycin-tetracycline therapy is currently held by many to be the treatment of choice. One method of management consists in giving 500 mg. of a tetracycline orally every six hours and 1 gm. of streptomycin intramuscularly every twelve hours daily for two weeks. Some investigators give a lesser amount of streptomy-

cin and administer the tetracycline for a longer period while others give only a tetracycline for a longer period.

Vibriosis. Infection due to Vibrio fetus resembles brucellosis in its pathogenesis and symptomatology and appears best managed by a regimen similar to that used in brucellosis, namely, streptomycin-tetracycline therapy.

Pasteurella. Tularemia is one of the few diseases in which streptomycin seemingly can be employed alone with excellent results. The tetracyclines also are effective, and in cases of severe infection they have been given conjointly with streptomycin. Kanamycin and chloramphenicol also are efficient antitularemic drugs but their use is avoided because of their potential untoward effects. The same drugs have been effective in plague, but in infections due to certain other Pasteurella organisms, such as P. multocida (P. septica), penicillin G is recommended. In addition to causing bacteremia, P. multocida may cause other infections such as cellulitis, osteomyelitis, meningitis, and pneumonia which appear to reflect contact with animals. In patients allergic to penicillin, erythromycin or a tetracycline is recommended against P. multocida.

Haemophilus. In serious infections due to H. influenzae, such as meningitis, invasion of the bloodstream is not infrequent. Tetracycline-streptomycin has been effective, but ampicillin or chloramphenicol probably is to be preferred, especially in rapidly progressive infections.

Achromobacter. Organisms of the Achromobacter family infrequently cause bacteremia, but tetracycline-streptomycin has been effective. In certain cases, in vitro susceptibility tests may indicate a more pertinent regimen.

Mimeae. In bacteremia due to the Mimeae group, that is, Mima polymorpha or Herrellea vaginocola, combined use of a tetracycline plus kanamycin, polymyxin B, or colistin is recommended when treatment is begun before in vitro susceptibilities are reported.

Clostridia. Bacteremia due to organisms of the genus Clostridium may occur in patients with underlying hematologic disease and in patients with neoplastic disease, but the majority of reported cases of clostridial bacteremia have been associated with postabortal or puerperal sepsis.

General supportive care, including blood transfusions when indicated, and the administration of antibiotics and polyvalent gas–gangrene antitoxin have been recommended for management of clostridial bacteremia. Penicillin G in massive doses appears to be the antibacterial agent of choice, but some investigators believe that a tetracycline may give additional benefit. Therapy with antimicrobial agents or antiserum is not regarded as a substitute for surgical extirpation of a septic focus. Placing the patient in a hyperbaric chamber in an attempt to increase the amount of oxygen in the tissues, particularly the oxygen in physical solution, may be essential and may obviate the need for some of the procedures previously recommended. Exposure to oxygen at three atmospheres of pressure for two-hour sessions appears to be generally safe and effective in a considerable number of cases.

Bacillus anthracis. Several antibacterial agents including penicillin G, erythromycin, a tetracycline, and sulfonamides have been effective in infections due to B. anthracis. Because penicillin G is bactericidal and less toxic than a tetracycline or a sulfonamide, it may be the agent of choice if the patient has not previously had an allergic reaction to the drug.

Listeriosis. Penicillin G, a tetracycline, or erythromycin has been effective in infections due to Listeria monocytogenes. Whether or not addition of streptomycin enhances the activity of penicillin G or a tetracycline is disputed. Although sometimes complicating neoplastic disease and diabetes mellitus, listeriosis has a propensity to occur during pregnancy. Usually the disease is seen in the infant and the mother is asymptomatic or has had an influenza-like illness. If alive at birth, infected infants usually give evidence of meningitis and bacteremia; in surviving infants, residua such as hydrocephalus are common.

Polymicrobic bacteremia. Polymicrobic infections are most likely to occur in bacteremia due to Bacteroides organisms and in infections of the blood due to S. faecalis. Since polymicrobic infections are of higher incidence in patients having hematologic disease, another organism also is not infrequently isolated simultaneously in such patients who have Pseudomonas bacteremia.

In treatment of polymicrobic bacteremia, several drugs may be given simultaneously, for example, a tetracycline in Bacteroides infection and, concomitantly, penicillin G and streptomycin against streptococci. Also, in addition to penicillin G and streptomycin administered in enterococcal bacteremia, one may add a tetracycline or chloramphenicol against E. coli or Klebsiella-Aerobacter organisms. Since it is difficult to say which organism is most important in polymicrobic bacteremia, one gives broad coverage. Perhaps ampicillin and cephalothin will have special application in selected polymicrobic infections.

Adjunctive therapy of bacteremia. In addition to antibacterial therapy which is essential for eradication of these infections, there are adjunctive or supportive measures that appear to warrant brief mention. Replacement of blood may be required in patients who have bleeding lesions of the gastrointestinal tract or underlying hematologic diseases. Fluid and electrolyte deficits may require correction; however, overadministration of fluids, especially saline solution, during a period of oliguria can cause death from embarrassment of cardiac function or from pulmonary edema. As a generalization, fluids can be given cautiously as long as the central venous pressure is low or normal, that is, 5 to 15 cm. of water.

Surgical intervention may be of therapeutic aid when a primary or a metastatic site of infection must be drained. The gallbladder, middle ear, or an obstructed portion of the urinary tract may become a nidus of infection from which the blood is reseeded with organisms, and nonsurgical treatment may prove inadequate. Metastatic suppuration requiring surgical attention is most likely to complicate bacteremia due to S. aureus, S. pyogenes, or Bacteroides organisms.

Bacteremic shock is most likely to complicate staphylococcemia, meningococcemia, and bacteremia due to gram-negative bacilli.[16] Controversy exists regarding the precise management of bacteremic shock, but measures that are frequently employed are mentioned, at least for the sake of completeness. Although any outline of therapy is controversial, different regimens are being employed in several medical centers and comparative data will become available. It is well to recall that symptomatic procedures are designed only to maintain the patient's life until antibiotic therapy can control the overwhelming infection which is the essential cause of his vascular collapse. Perhaps more important than the investigation of additional supportive measures are the recognition of the potential candidate and the monitoring of his temperature, blood pressure, and urinary output so that early diagnosis and antibiotic treatment of the complication are possible.

Vasopressors have been used in an attempt to restore circulatory homeostasis by reinforcing the natural bodily reaction to the complication, to forestall so-called irreversible shock, and to restore circulation before ischemia has damaged the kidneys and produced renal shutdown. As will be noted later, a current question is whether vasodilators[17] should be used rather than vasopressors. The three vasopressor agents that appear to be most commonly used at present are metaraminol (Aramine®), levarterenol (Levophed®), and angiotensin (Hypertensin®). If a Y-tube apparatus is used to deliver vasopressor drugs given intravenously, administration of the vasopressor can be discontinued periodically so that only a minimal amount is given but the solution remains immediately available if hypotension recurs. It probably is unnecessary to raise the systolic blood pressure above 100 mm. Hg in a previously normotensive patient.

Metaraminol can be given intramuscularly but preferably is given intravenously in amounts of 100 to 250 mg. in 500 to 1,000 ml. of fluid. Some experimental findings indicate that release of levarterenol from sympathetic nerves has a major role in the action of metaraminol. If this is correct, when refractoriness to metaraminol is suspected, levarterenol should be substituted. Since reserpine may deplete stores of levarterenol, metaraminol also may not be advisable in the management of patients who recently have received such catecholamine-depleting drugs.

Levarterenol is given intravenously in amounts of 4 to 16 mg. in 1 liter of 5% dextrose in water. When levarterenol is employed, the site of infusion and the course of the vein infused should be inspected frequently for evidence of extravasation, such as a hard, cold, blanched area of skin. If ischemia is noted, necrosis may be prevented or minimized by stopping the infusion promptly and infiltrating the region with adrenergic blocking agents. Injection of blanched areas with either 5 to 10 mg. of phentolamine (Regitine®), with or without 150 turbidity units of hyaluronidase, or 5 mg. of piperoxan (Benodaine®) contained in 10 to 20 ml. of saline solution is recommended; injection is made with a long 22-gauge needle and is directed to all parts of the blanched area. Protection against necrosis of the skin after extravasation of levarterenol is said to be possible by the addition of 5 to 10 mg. of phentolamine or 10 to 20 mg. of heparin to each liter of solution containing the agent. Phentolamine used in this manner is said not to reduce the pressor effect of levarterenol despite antagonizing its local vasoconstrictor action. Neither metaraminol nor levarterenol should be used in patients to whom cyclopropane anesthesia or other potentially cardiac-sensitizing agents recently have been administered.

Angiotensin has given encouraging results in maintaining the patient's blood pressure during an episode of bacteremic shock. A vial of angiotensin contains 2.5 mg. of the drug and, when given intravenously, usually 1 to 4 vials are

added to a liter of fluid; as many as 10 vials have been given in a twenty-four-hour period. Angiotensin is considered to have stronger action on the visceral blood vessels in contradistinction to levarterenol, which chiefly affects blood vessels of the muscles. Unlike levarterenol, angiotensin is said not to induce dependence or reflex hypotension on its withdrawal. Apparently, certain patients not responding well to levarterenol have responded to angiotensin and, since the converse also has occurred in some, a trial of both drugs may be required.

Largely on an investigational basis, adrenergic or sympathetic blocking agents, such as phenoxybenzamine (Dibenzyline®) in amounts of 1 mg. per kilogram of body weight administered by slow intravenous drip, have been used in patients in bacteremic shock in the hope of inducing more normal redistribution of the blood flow which, in turn, will result in a restoration of the blood pressure to normal. Evaluation of these agents is being made because some findings, such as pallor, sweating, and tachycardia, are signs of vasoconstriction and overactivity of the sympathetic nervous system. Therefore, it has been theorized that dilatation of blood vessels, rather than their further constriction by vasopressor agents, may be required to induce local redistribution of blood and speeding up of flow with relief of visceral pooling. It should be noted that little data on human subjects and their response to sympathetic blocking agents are available. The majority of conclusions regarding their effects in bacteremic shock are based on canine experiments, and these are not automatically transferable to man. Also, one should be aware that drugs such as phenoxybenzamine may further reduce the blood pressure[18] and require administration of blood or a blood substitute. Isoproterenol (Isuprel®), which is used in cardiogenic shock, may cause peripheral vasodilatation and is being reevaluated in bacteremic shock. Mephentermine (Wyamine®) produces a result similar to that of isoproterenol. Other vasodilators which are being investigated include phentolamine, trimethaphan (Arfonad®), and hydralazine (Apresoline®).

Oxygen therapy has been recommended to counteract tissue anoxia resulting from the reduced arterial blood flow, but the logic of its use is disputed. Hyperbaric oxygenation does not appear to have application to the management of bacteremic shock at this time.

An airway is provided if necessary.

Digitalization may be carried out if the central venous pressure remains above 15 cm. of water and particularly if corroborative evidences of heart failure, such as pulmonary edema, are detected.

Mechanical measures, such as elevating the foot of the patient's bed, application of an abdominal binder, and bandaging of the lower extremities, are designed to increase venous return to the heart and, consequently, increase cardiac output. However, there are no exact data which indicate that these measures are of substantial benefit.

If hypothermia is not used, the patient may be kept comfortably warm, but overheating is to be avoided. Overheating may induce dilatation of superficial vessels and further increase the capacity of the vascular bed. The exact application of hypothermia in bacteremic shock remains unclear.

In patients suffering from renal shutdown, usually the first sign of reversibility is reappearance of excretory function within ten to twelve days. Excretory function reappears before regulatory function, and death with progressive azotemia and acidosis remains possible at this stage. Hemodialysis has contributed significantly to maintaining life and reducing morbidity during the oliguric phase of renal shutdown. It should not be reserved as a last desperate measure; its failure to prevent death may reflect belated use. Persistent oliguria may necessitate repeated hemodialysis.

In selected cases wherein oliguria occurs and it is not certain if renal shutdown is responsible, 25 gm. of mannitol are given intravenously. In some of these cases, diuresis follows shortly and consideration of hemodialysis can be deferred.

Use of morphine, meperidine (Demerol®), barbiturates, and other depressants is best avoided.

Some investigators note a favorable response to the use of adrenocorticosteroids in cases of bacteremic shock despite the fact that these patients, even when moribund, show no abnormality of the secretory function of the adrenal gland.[19] We are not persuaded that use of these agents is mandatory on the fragile basis of comparing mortality rates in reported series wherein such steroids were employed. Actually, such comparison might lead one to conclude that patients who have received this type of steroid in attempts to control shock have a higher mortality rate. Among objections to steroid therapy are impaired immune response, increased susceptiblity to upper gastrointestinal bleeding, and fatal staphylococcal superinfection. We have limited the administration of adrenocorticosteroids in nonpharmacologic doses to those patients who had been receiving them before the

occurrence of bacteremia and bacteremic shock.

Also, we have not used blood or plasma in the definitive management of bacteremic shock in our patients because there appears to be no decrease in blood volume but rather stagnation and faulty redistribution of it. It is recognized, of course, that replacement of blood may be required in patients having bleeding lesions of the gastrointestinal tract or underlying hematologic diseases. We are currently investigating the fairly rapid intravenous infusion of 500 mg. of dextran[20] and, in selected cases, such infusion may restore the blood pressure and make unnecessary the use of a vasopressor or vasodilator. Low molecular dextran (40,000) is given in amounts of 500 ml. over thirty to one hundred twenty minutes at twelve-hour intervals.

Heparinization to counteract intravascular coagulation during experimental bacteremic shock has been advocated but its value lacks clinical confirmation. In this regard, it is to be recalled that numerous other regimens, including testosterone, aldosterone, serotonin antagonists, chlorpromazine, and even antihistaminics, have been described as being helpful in experimental animals, but this does not automatically assure their efficacy in the human subject.

CONCLUSIONS

With the advent of effective antibacterial agents, the incidences of streptococcal and pneumococcal bacteremia have decreased considerably. This empty ecologic niche is being filled by a progressively increasing incidence of bacteremia due to gram-negative bacilli. In the management of patients having bacteremia, it is necessary that one be acquainted with the underlying or predisposing diseases and forms of treatment which may influence the type of infecting pathogen, hinder the patient's recovery from the infection, or require special therapeutic attention. The many sides of bacteremia, such as polymicrobic cause or causation by organisms requiring anaerobic culture techniques, and such complications as bacteremic shock also indicate the need for special knowledge on the part of the physician caring for patients with such infections. A primary nidus of infection, from which the bloodstream has been seeded, or a site of metastatic suppuration may require surgical intervention. "Cure" in bacteremia is determined only by the patient's progress from both clinical and laboratory points of view after cessation of antibacterial therapy. Careful analysis of individual cases of bacteremia and the use of common sense in application of antibacterial drugs and adjunctive therapy are required.

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Oesophagopleural Fistula:

A Complication of Lobectomy, Report of an Unusual Case

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THE CAUSES OF ESOPHAGEAL perforation and subsequent fistula formation have been clearly defined, and instrumental perforation is the commonest.[1–7]

In most instances, whether the perforation is due to trauma from an instrument, a swallowed foreign body, or the result of postemetic spontaneous rupture, there is a good chance of recovery when the lesion is readily recognized and treated. Failure to make the diagnosis and institute appropriate treatment leads to severe complications and a high mortality rate.[8–11]

The development of an esophageal fistula following lobectomy is a rare condition. A case is reported here in which such a fistula developed after a lobectomy was carried out in the course of treating an empyema. It is presented to emphasize the possibility of the occurrence of this complication after this type of surgery.

CASE REPORT

A 37-year-old woman was admitted to the Central Chest Hospital on Sept. 16, 1963. Six years previously she had had an illness with productive cough, fever, and malaise. She was admitted to another hospital where a diagnosis of lung abscess was made. However, she refused surgical treatment and went home. In the intervening years her general condition had gradually deteriorated but without any acute exacerbation of symptoms. The cough persisted and she occasionally had bouts of fever.

On admission her chief complaint was a cough, productive of 100 to 200 cc. of sputum daily. There was no history of hemoptysis or chest pain. Since her previous admission she had lost approximately 10 kg. of weight.

She was a thin, pale, undernourished, and toxic looking patient. Temperature was 37.2° C., pulse was 92 per minute, respiration was 24 per minute, and blood pressure was 100/58 mm. Hg. Significant findings were in the chest where there was dullness of the right lower chest posteriorly and moist rales were audible over the right lower and middle lobes. Examination of the other systems was noncontributory.

LABORATORY FINDINGS

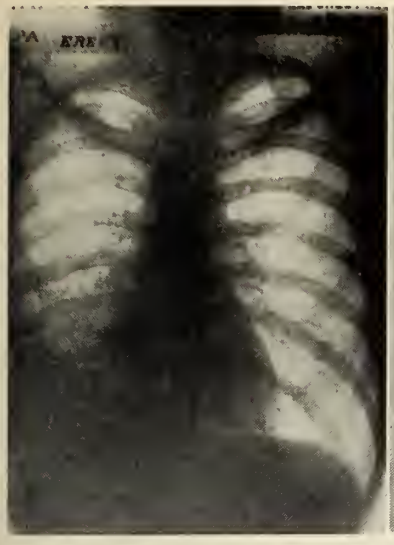
Hemoglobin was 9 gm., red blood count was 2.74 m., white blood count was 9,250, polymorphocytes 27%, and lymphocytes 73%. Erythrocyte sedimentation rate was 125 mm. per hour. Urinalysis was within normal limits. Sputum was negative for acid fast bacilli on smear and no tubercle bacilli were grown after culture. The predominant organism on culture was P. aerobactor. Other laboratory findings were within normal limits.

PROGRESS

A diagnosis of chronic lung abscess was made and the treatment was started with 2 million units of penicillin G. daily. Her general condition and X-ray findings subsequently showed much improvement. The residual lesion and bronchial deformities of the right middle lobe and right lower lobe were clearly demonstrated by the bronchography. The resection of residual disease was indicated.

On Dec. 2, 1963, she underwent removal of the right middle and lower lobes. Because of the extent and nature of the adhesions between the lung and the posterior mediastinum much dissection was needed to free the lobes. At no time was it thought that the esophagus had been

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Figs. 1 *(left)* and 2 *(right)* Posteroanterior and lateral views of chest preoperatively, note haziness of residual disease at right base.



Fig. 3. Bronchogram clearly shows localization of disease at right middle lobe and right lower lobe.

injured although its fibers were clearly seen close to the inferior pulmonary vein.

The patient's progress postoperatively was good for the first twenty-four hours, but then the drainage from the lower chest tube became turbid and grew E. coli on culture. A chest X-ray at this time did not show any significant changes. During the next forty-eight hours the amount of chest drainage increased to 1,600 cc. per day and became frankly purulent. However, her general condition at this time was good except the elevation of temperature exceeding 104° F. Streptomycin sulfate (Streptomycin®), one-half gm. every 8 hours and penicillin G. sodium, 2 million units per day, were given and the patient continued to take fluids by mouth.

By the tenth postoperative day a large abscess cavity could be seen at the right base by X-ray. Therefore, under local anesthesia the eighth rib was partially resected, a large amount of necrotic debris was removed from the cavity, and a large bore tube inserted and connected to underwater seal drainage. Because of the unusual characteristic of drainage materials showing ingested food, the esophagopleural fistula was suspected and confirmed by barium swallowing. On Dec. 16, a second thoracotomy was carried out. The empyema cavity was thoroughly evacuated and

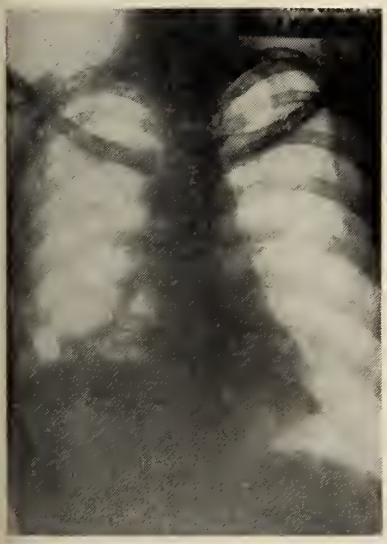
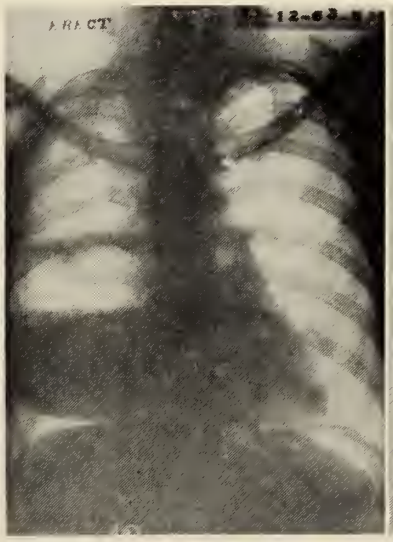
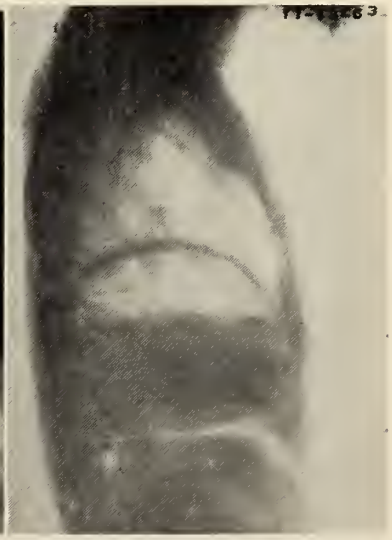
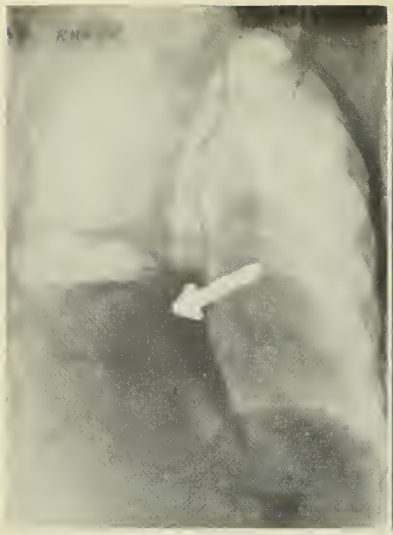


Fig. 4. Posteroanterior view of chest after right middle lobe and right lower lobe lobectomy.





Figs. 5 *(left)* and 6 *(right)*. Tenth postoperative day posteroanterior and lateral view of chest reveal large abscess cavity at right base.



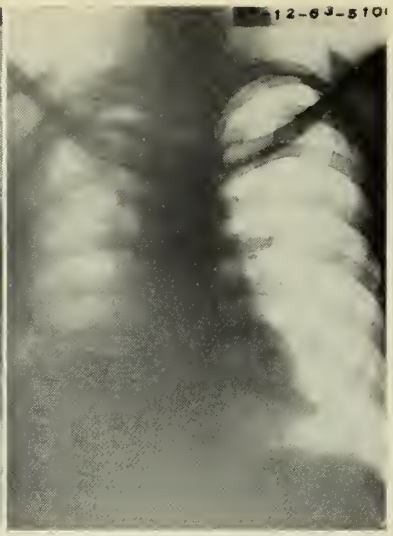


Fig. 7. Barium swallow shows opaque material entering abscess cavity (arrow).

Fig. 8. PA. Chest postrepair of esophagopleural fistula; note elevation of right dome of diaphragm without residual space.



Fig. 9. Barium swallow few weeks postrepair reveals only slight irregularity of esophagael wall (arrow) but no leakage is demonstrated.

completely excised. The esophagopleural fistula was found just below the region of the previous inferior pulmonary vein. An opening in the esophagus was about 1 cm. long. It was then isolated from the surrounding tissue and closed with interrupted sutures of silk in 3 layers. The edges of the fistula were firm and did accept sutures well.

Postoperatively, the patient was fed by a nasogastric tube, which remained in place until Jan. 13, when another barium swallowing revealed no leakage. Only slight irregularity at the site of repair was noted. The patient was thereafter fed by mouth without difficulty. The follow-up six months later showed the patient had gained 15 lb. and was able to swallow normally.

DISCUSSION

The esophagopleural fistula following pulmonary resection is indeed a rare condition. The significance of this case was the diagnosis proceeding correct and proper treatment. One should be suspicious of esophagopleural fistula by observing excessive drainage, forty-eight to seventy-two hours postoperatively, from the pleural cavity. The drainage rapidly becomes purulent. If there is any doubt concerning the diagnosis, further investigation, such as swallowing methylene blue or opaque material, would be helpful. Surgical exploration and repair of the fistula should be done promptly following the correct diagnosis. If there is a delay in the process of treatment and the drainage becomes purulent, the rib resection drainage is the procedure of choice. When the infection is under control, repairing the fistula must then be considered. After the fistula is closed, the patients can be fed by either nasogastric tube or gastrostomy. The tube feeding was satisfactory in this case.

SUMMARY

A case of esophagopleural fistula following pulmonary resection is presented. The diagnosis was made by demonstration of the methylene blue, and definite location of the fistula was seen by opaque media swallowing. The repairing of the fistula was performed two weeks postlobectomy with successful results. By March 1965 the patient was asymptomatic and could eat normally.

The authors wish to express their appreciation to Dr. Vai Chinachote, director of Central Chest Hospital, Nonburi, for his permission to report this case.

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Hyperglycemic Nonketotic Coma Induced by Peritoneal Dialysis

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THIS REPORT documents the progressive deterioration in the level of consciousness in a diabetic patient undergoing peritoneal dialysis. The loss occurred paradoxically in spite of progressive improvement of pulmonary edema, metabolic acidosis, hyperkalemia, and hyponatremia, for which the procedure was instituted. The development of coma appeared to be related to a precipitous rise in blood sugar which occurred during dialysis, and a subsequent return of consciousness followed lowering of the blood sugar levels.

This report is prompted because of the relative infrequency of significant hyperglycemia complicating peritoneal dialysis[1–3] and because this case most likely represents an instance of nonketotic hyperglycemic coma[4] induced by peritoneal dialysis.

CASE REPORT

A 26-year-old female with nephropathy, neuropathy, and retinopathy associated with diabetes entered the hospital on Jan. 17, 1966, with cough, anterior pleuritic chest pain, and swollen feet. The patient was acutely ill but alert and cooperative.

Examination revealed the following: blood pressure, 170/74; pulse rate, 132; respirations, 24; and temperature, 104° F. Examination of the lungs revealed bilateral basilar rales. Cardiomegaly, a tender, enlarged liver, and 3+ pitting edema in the lower extremities were noted. Diminished pain perception also was noted in the lower extremities. Neck veins were not distended. Pertinent laboratory data relating to this hospitalization are found in the table.

Treatment for left lower pneumonitis, congestive heart failure, and diabetes consisted of insulin and penicillin as well as bed rest. Within two days, the patient was afebrile and clinically improved.

On the morning of Jan. 20, the patient was found to have pulmonary edema. Rales were noted throughout both lung fields, and the patient was orthopneic, with tachycardia and a diastolic gallop rhythm. The pulmonary edema was not alleviated by the employment of rotating tourniquets, oxygen, morphine, and phlebotomy. Arterial hydrogen ion concentration was 7.2. Serum electrolyte determination (see table) reflected hyperkalemia, a depressed total CO_2 content, and significant hyponatremia despite correcting for the presence of hyperglycemia. A trace-positive test for ketonemia was detected. Peritoneal dialysis was instituted, with dialysate containing 1,500 mg. percent glucose, to treat pulmonary edema as well as to ameliorate acidosis, hyperkalemia, and hyponatremia. On clinical grounds, the metabolic acidosis was ascribed primarily to the presence of significant renal insufficiency rather than to diabetic ketoacidosis. Even when well, this patient's total CO_2 content ranged from 11 to 13 mEq. per liter, with a BUN of 40 to 50 mg. percent. In addition, the ketonemia was only faintly positive in contrast to the strongly positive reaction, requiring several dilutions, detected in diabetic ketoacidosis.

During the twenty-four hours of dialysis, a significant negative fluid balance was effected, together with reversal of the biochemical abnormalities (see table). No glucose was administered intravenously during this period. The blood sugar levels progressively increased despite employment of moderate doses of insulin, the highest value observed being 1,120 mg. percent seven hours after institution of dialysis. Significant alteration in the patient's level of consciousness was observed during the procedure. Before dialysis, the patient was able to respond to questions and commands. In sharp contrast, despite chemical improvement during dialysis, she became progressively mentally obtunded and had generalized twitching movements. No localizing signs or abnormal reflexes were elicited on neurological examination. Dialysis was terminated as rapidly as possible because of deterioration in the patient's level of consciousness.

The comatose state, which correlated temporally with significant hyperglycemia, paradoxically, occurred with progressive improvement of acidosis, hyperkalemia, and hyponatremia. It was assumed that amelioration of the hyperglycemia and, hopefully, improvement in the patient's depressed mental state could be achieved more readily by discontinuing dialysis against a gradient of 1,500 mg. percent glucose.

The patient remained semicomatose during the immediate postdialysis period. Administration of small amounts of regular insulin reduced the elevated blood sugar levels. A lumbar puncture was performed nine hours after termination of dialysis because of continuing semicoma. Cerebrospinal fluid was normal. By the twenty-fifth postdialysis hour, the patient was fully awake and responsive. She had no recollection of the events during the previous fifty hours.

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LABORATORY DATA AND TREATMENT SCHEDULE

Date	Clinical state	Osmol. 2° to hyperglycemia*	Calcd. ECF† osmol.	Electrolytes: Na	K	Cl	CO_2	BUN	Blood glucose	Serum acetone	pH‡	Urinalysis: Specific gravity	Protein	Glucose	Acetone	Hematology: WBC	Hematocrit	Therapy
Jan. 17	Coughing, anterior pleuritic chest pain, fever		276	133	5.0	108	13	42								9,850	21	5 units regular insulin
Jan. 18, 5:30 A.M.	Temperature down to 101° F.		286	138	5.0	112	12		56					Neg.	Neg.			
12:00 Noon	Cough persisting								132									
6:00 P.M.								52	618									15 units regular insulin
Jan. 19	Temperature, 98° F.; complaints of orthopnea; weight, 48 kg. (106 lb.)								496 232					1+	Neg.	8,300	20	
Jan. 20, 6:30 A.M.	Increasing dyspnea; weight, 51 kg. (112 lb.)											1.012	3+	3+	Trace			
9:00 A.M.	Tachycardia and diastolic gallop	22	248	108	7.4	88	4.0	52	496	Trace	7.20							15 units regular insulin
1:30 P.M.				115	7.2	86	6.5	46										
Dialysis started																		
3:00 P.M.		43	285	116	5.6	90	11.0	43	865					4+	Trace			20 units regular insulin
8:00 P.M.	Twitching movements	57	317	125	5.0	98	9.2		1,120					4+	Neg.			
Jan. 21, 7:00 A.M.	Sensorium obtunded; twitching movements; weight, 47.3 kg. (104 lb.)	46	320	132	5.4	96	15	28	926		7.37							15 units lente insulin
11:30 A.M.	Incoherent speech	40	316	133	4.5	106	21	23	820									20 units regular insulin
1:00 P.M.	Patient obtunded																	
Dialysis stopped																		
3:30 P.M.		45	303	124	3.7	106	23	24	900					2+	Neg.			20 units regular insulin
8:00 P.M.	Obtunded; lumbar puncture	12	292	135	4.1	101	21	25	312									
Jan. 22, 12 P.M.	Responding. No recent memory of events of past 48 hours		284	137	4.5	101	25	36	93					Trace	Neg.			

*Calculated osmolarity $= \frac{\text{Observed blood sugar} - 100 \text{ mg. percent} \times 10}{180}$

†Calculated extracellular fluid osmolarity = Serum Na × 2 + 10 + Osmol. of excess glucose

‡Arterial pH

The remainder of the hospital course was uneventful except for several bouts of hypoglycemia. She was discharged on Jan. 27 and was followed in the outpatient department.

DISCUSSION

The indications for peritoneal dialysis in this patient included: [1] refractory pulmonary edema, [2] hyperkalemia, [3] metabolic acidosis, and [4] hyponatremia. The metabolic acidosis and hyperkalemia were ascribed primarily to the presence of chronic renal disease, most likely diabetic glomerulosclerosis. For the reasons alluded to earlier, the faintly positive test for serum ketone was not considered to be unequivocal evidence for the presence of diabetic ketoacidosis. In addition, relatively small amounts of insulin were utilized in reducing the blood sugar, in contrast to the larger doses of insulin usually required to treat diabetic ketoacidosis. It is recognized that lactic acidosis—not of the phenformin type[5]—can occur in patients with diabetes.[6] Although this remains a possibility on clinical grounds, the greater likelihood is that the metabolic acidosis observed in this patient reflected significant impairment of renal function.

Although chemical and cardiovascular improvement was being effected, the patient's mental state progressively deteriorated, accompanied by generalized twitching movements. The possibility of the "dialysis disequilibrium syndrome"[7–9] was considered but dismissed for the following reasons: [1] the rate of urea removal is approximately four times slower with peritoneal dialysis than with hemodialysis[2] and [2] the predialysis BUN (42 mg. percent) was well below the range usually associated with the disequilibrium syndrome. However, this possibility could not be excluded entirely.

Maher and Schreiner[1] observed blood sugar levels greater than 400 mg. percent in 5 out of 228 peritoneal dialyses performed in 119 patients. An instance of transient blindness is reported in association with hyperglycemia in one patient, and the relationship of coma to significantly increased blood sugar levels is mentioned. It would appear then that the most likely explanation for CNS signs and symptoms observed in this patient during dialysis was the significant and acute rise in blood sugar concentration. The relationship between significant hyperglycemia and onset of coma is well documented clinically.[5] In many of the reported cases of nonketotic hyperglycemic coma, the extent of fluid depletion has been profound, and massive fluid replacement has been required in therapy. Administration of large increments of fluid was not required in our patient for two reasons: [1] the preexisting overloaded circulatory system and [2] the relatively short elapsed time from detection of significant hyperglycemia to correction of the elevated levels. The CNS response to hypertonicity induced by hyperglycemia can take the form of focal seizures. Seven cases have been documented in which focal seizures responded poorly to anticonvulsant therapy but favorably to rehydration and insulin.[10]

Experimental data indicate that increasing levels of blood glucose in dogs is correlated with progression in CNS signs and symptoms beginning with restlessness noted at levels of 2,000 mg. percent, advancing to tonic-clonic convulsion at 3,000 mg. percent, and terminating in death when glucose concentration attained 3,700 to 3,800 mg. percent. Morphologically, lesions were present in the meninges and the brain.[11]

The manner in which hyperglycemia affects cellular function is not fully understood. The most likely explanation is that of cellular dehydration, that is, the efflux of cell water into the extracellular space in response to the hyperosmoticity induced by significant hyperglycemia.[12] Alloxanized, diabetic rats[13] exhibit significant cellular deficits of potassium and water accompanied with hyperglycemia and acidosis. Similarly, cellular losses of both water and electrolytes have been appreciated in diabetic ketoacidosis.[14] However, these studies do not delineate precisely the influence which hyperglycemia per se exerts on cell composition because of the concomitant renal response to the osmotic load of glucose, namely, the sweeping out of both water and electrolytes by way of the urine.

It is important to emphasize that, in yet another clinical instance of cellular dehydration, namely, hypernatremic dehydration, CNS signs and symptoms ranging from stupor to coma and convulsions are encountered.[15–17] The concurrence of hypernatremia and subdural effusions or bleeding has been observed clinically[18] as well as under experimental circumstances.[19]

A fatal outcome has been reported in association with a blood glucose concentration of 3,400 mg. percent which occurred during hemodialysis utilizing a bath glucose concentration of 7,000 mg. percent. Hyperglycemia was accompanied by confusion and disorientation.[20] This case may represent the clinical counterpart of Wierzuchowski's experimental observations in which dogs died when blood glucose levels attained 3,700 to 3,800 mg. percent.

The present observations indicate that progressive loss of consciousness during peritoneal dialysis, especially in patients with diabetes,

should suggest the presence of significant hyperglycemia. Early recognition and treatment of hyperglycemia may contribute significantly toward effecting a favorable outcome in a potentially life-threatening situation, for the overall mortality in patients with nonketotic hyperglycemic coma approximates 40%.[21]

SUMMARY

The onset of progressive stupor and twitching in a diabetic patient undergoing peritoneal dialysis was accompanied by significant hyperglycemia. Return to a normal state of consciousness appeared to correlate in time with reduction of the hyperglycemia. The sequence of events can best be accounted for by postulating cellular dehydration secondary to the extracellular hyperosmoticity exerted by the elevated blood glucose concentration. This case is reported to emphasize the point that, in diabetic patients undergoing dialysis, the progressive loss of consciousness occurring at a time when clinical improvement is anticipated should alert the clinician to the presence of significant hyperglycemia.

The author wishes to thank Drs. S. Papper and A. N. Longfield for helpful comments and criticisms and Miss Naomi Hagan for secretarial assistance.

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Tropical Pulmonary Eosinophilia: A Clinical Study

R. K. NARANG, M.D.

Kanpur, India

Eosinophilia may be a feature of many chest diseases or of many generalized diseases in which a pulmonary lesion is a predominant manifestation. This paper is concerned with a well-defined group that Ball has called "tropical pulmonary eosinophilia."[1] Attention was first drawn to this condition by Weingarten[2] in India and the condition is sometimes called Weingarten's syndrome. The syndrome is often confused with many eosinophilic lung conditions like Löeffler's syndrome or polyarteritis nodosa and many chronic diseases like bronchitis, asthma, pulmonary tuberculosis, and fungus diseases of the lung. For this reason, the author is prompted to draw attention to various manifestations and diagnostic features of the disease.

MATERIAL AND METHODS

Selection of cases. The present study consisted of patients who fulfilled the following criteria: [1] an absolute eosinophil count of 2,000 or more per cubic millimeter, [2] repeated stool examinations negative for any helminthic infection, and [3] three samples of blood taken on successive nights negative for microfilaria bancrofti.

These investigations were necessary to exclude parasite infection of intestines and filariasis, conditions associated with eosinophilia and common in India. Pulmonary filariasis, described by Rifkin and Erberhard,[3] is especially likely to be confused with tropical pulmonary eosinophilia.

Plan of investigation. The clinical history included a record of age, sex, and occupation; details of various symptoms; history of past illness with special reference to any evidence of allergy or parasitic infections; and family history.

A thorough general and systemic examination was done. Special note was taken of the presence of lymphadenopathy, hepatomegaly, splenomegaly, or skin lesions.

Laboratory investigations included a total leukocyte count, an absolute eosinophil count using the technique described by Dacie,[4] the erythrocyte sedimentation rate (Wintrobe), and a skiagram of the chest, posteroanterior view.

OBSERVATIONS AND RESULTS

Clinical findings. The study consisted of 77 men and 11 women, ranging in age from 12 to 61 years. The majority of patients (91%) were in the 15-to-44 age group (Table 1).

The duration of illness reported by the patient when first under observation was variable (Table 2), the shortest period being ten days and the longest fourteen years. However, in the majority of patients (80.7%) it was under six months. Most of the patients delayed seeking admission because they came from distant villages where they were under the treatment of Ayurvedic practitioners. Where the history was of long duration, the patients had been treated mistakenly for bronchitis, pulmonary tuberculosis, or asthma.

The commonest symptom in all patients (Table 3) was cough occurring in paroxysms of a few seconds and productive of slight or no sputum. In 93.2% a feeling of suffocation and breathlessness was usually present after each bout of coughing. A dull substernal pain was the next common symptom, followed by hemoptysis and

TABLE 1

AGE INCIDENCE

Age groups (in years)	*No. of patients*	*Percentage of total*
0 to 4	—	—
5 to 15	2	2.2
15 to 24	21	23.8
25 to 44	59	67.2
45 to 64	6	6.8
65 and over	—	—
Total	88	100

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TABLE 2

DURATION OF ILLNESS

Duration	*No. of patients*	*Percentage of total*
Under 6 months	71	80.7
6 to 12 months	14	15.9
Over 12 months	3	3.4
Total	88	100

low grade fever with a maximum of 100° F. Hemoptysis consisted only of slight streaks of blood; no patient had frank hemoptysis. Loss of weight was present in three patients who had been ill two years, three years, and fourteen years, respectively, when they were first examined.

The degree of incapacitation (Table 4) was assessed according to the presence or absence of breathlessness. Patients with slight severity had cough only, while patients with severe symptoms had constant dyspnea. No case of status asthmaticus was noted.

Allergy was found in 3 patients who gave a past history of urticaria. Two patients had a family history of urticaria and one a family history of bronchial asthma.

Physical examination (Table 5) showed that only 67.1% of the patients had physical signs in the chest. These included harsh vesicular breathing and prolonged expiration with adventitious sounds, mainly ronchi, wheezing, and crepitations. Clubbing or cyanosis was not noted in any patient.

Only the respiratory findings were abnormal. Hepatomegaly, splenomegaly, lymphadenopathy, and cutaneous lesions were conspicuous by their absence.

Radiological findings (Table 6). Increased striations were the most frequent finding. The striations, which extended almost to the periphery of the lung fields, were most marked in the lower and middle zones. In addition, a varying degree of pulmonary mottling was also present in 32.2% of patients. The mottling consisted of numerous discrete, soft-rounded shadows with ill-defined margins, usually of pin-head size, scattered throughout the lung fields; they were most marked in the base and mid-zone areas, lessening in intensity from below to upward and leaving the apexes clear. In 6 patients, bilateral hilar lymphadenopathy was superadded to this picture. One patient who had been ill fourteen years had typical emphysematous changes present in both lungs.

Hematological findings. Leukocytosis was present in all the patients (Table 7). The lowest leukocyte count was 14,150 per cubic millimeter and the highest was 82,000 per cubic millimeter. The majority of the patients had a count under 30,000 per cubic millimeter.

The lowest eosinophil count noted in the present series was 2,000 per cubic millimeter and the highest was 49,500 per cubic millimeter (Table

TABLE 3

FREQUENCY OF VARIOUS SYMPTOMS

Symptoms	*No. of patients (Total, 88)*	*Percentage of total*
Cough only	6	6.8
Paroxysms of cough with breathlessness	82	93.2
Chest pain	30	34.1
Hemoptysis	22	25.0
Fever	14	15.8
Loss of weight	3	3.4

TABLE 4

SEVERITY OF INCAPACITY

Degree of severity	*No. of patients*	*Percentage of total*
Slight	6	6.8
Moderately severe	59	67.1
Severe	23	26.1
Status asthmaticus	—	—
Total	88	100

TABLE 5

INCIDENCE OF PHYSICAL CHEST SIGNS

Physical chest signs	*No. of patients*	*Percentage of total*
No signs	29	32.9
Ronchi only	33	37.5
Wheeze only	5	5.6
Crepitations only	—	—
Ronchi, wheeze, and crepitations	21	24.0
Total	88	100

TABLE 6

INCIDENCE OF RADIOLOGICAL FINDINGS

Radiological findings	*No. of patients*	*Percentage of total*
Normal findings	3	3.6
Increased striations only	56	63.0
Increased striations and mottling	22	25.0
Striations, Mottling and Hilar lymphadenopathy	6	7.2
Emphysematous changes	1	1.2
Total	88	100

8). No relation was found between the duration and severity of symptoms and the eosinophil count.

A high erythrocyte sedimentation rate was found in only 67.1% of the patients (Table 9). For this study, an E.S.R. (Wintrobe) reading below 20 mm. for the first hour was taken as normal.

DISCUSSION

Cough, occurring in paroxysms, more severe at night, and associated with breathlessness after each bout, was the commonest complaint. This history was so characteristic that in a large number of patients a provisional diagnosis of tropical pulmonary eosinophilia was possible on this history alone. The diagnosis was confirmed on the finding of a massive eosinophilia in the peripheral blood. The minimum diagnostic level of count is not agreed upon by other investigators.

TABLE 7

RANGE OF TOTAL LEUKOCYTE COUNT

Leukocyte count per cubic millimeter	*No. of patients*	*Percentage of total*
Under 10,000	—	—
10,000 to 29,000	49	55.6
30,000 to 49,000	31	34.8
50,000 to 69,000	7	8.4
70,000 to 89,000	1	1.2
Over 90,000	—	—
Total	88	100

TABLE 8

RANGE OF ABSOLUTE EOSINOPHIL COUNT

Eosinophil count per cubic millimeter	*No. of patients*	*Percentage of total*
2,000 to 3,000	13	14.7
3,000 to 5,000	20	22.7
6,000 to 10,000	47	53.0
11,000 to 15,000	6	7.2
16,000 to 20,000	1	1.2
Over 20,000	1	1.2
Total	88	100

TABLE 9

RANGE OF E.S.R.

E.S.R. range	*No. of patients*	*Percentage of total*
Under 20 mm.	29	32.9
20 to 49 mm.	45	51.1
Over 50 mm.	14	16.0
Total	88	100

An eosinophil count of 2,000 per cubic millimeter[5] to 5,000 per cubic millimeter[6] has been taken as the dividing line between the syndrome of tropical pulmonary eosinophil and cosinophils due to other causes. In the present series, an eosinophil count of 2,000 or more per cubic millimeter has been taken as diagnostic, because all such cases have the typical clinical features of the syndrome and, according to the experience of the present worker, respond uniformly to treatment with diethylcarbamazine or to arsenic. Another important diagnostic point stressed by Crofton and associates[7] is the presence of leukocytosis. A moderate degree of leukocytosis was present in all the patients in the present study and was apparently due to eosinophilia. The author thinks that along with eosinophilia, a leukocytosis is a must for the diagnosis of tropical pulmonary eosinophilia. Presence of eosinophilia with a normal total leukocyte count calls for a search for some other cause of cosinophilia.

Extrapulmonary manifestations of the syndrome have been reported by various workers. Thus, Vytilligan[8] found hepatomegaly in all of his thirty-seven cases; Chaudhari,[9] Nath and Pandeya,[10] and Joseph[11] also describe hepatomegaly in their reports. Splenomegaly has been reported by Weingarten,[2] Chaudhari,[9] and Nath and Pandeya.[10] Generalized lymphadenopathy has been observed by Chaudhari,[9] Nath and Pandeya,[10] and Joseph.[11] Cutaneous manifestations in the form of nodular eruptions have also been described.[10,11] These manifestations were absent in the present series.

The disease affects patients in the best years of their lives; in the present report 91% were in the age group 15 to 44 years. Men accounted for 87.5% of all cases. The predominance of men, also noted in other reports, may well be due to a higher number of men patients seeking medical advice.

The characteristic radiological finding was increased striations with or without varying degrees of pulmonary mottling. Hilar lymphadenopathy, described by Ball,[1] was found in six patients. This appearance is likely to be confused with other diffuse diseases of the lung. However, in India tropical pulmonary eosinophilia is one of the commonest causes of this X-ray picture. Islam and Nurulhaq[12] have described a case of tropical pulmonary eosinophilia with cavitation.

It is surprising how often patients are mistakenly treated for bronchitis, asthma, or even pulmonary tuberculosis. A careful history taking, a routine eosinophil count, and, above all, an

awareness of the condition is all that is necessary to diagnose an easily curable disease.

SUMMARY

Clinical, radiological, and hematological changes in 88 cases of tropical pulmonary eosinophilia are described. The relevant literature also is discussed.

The author thanks Prof. K. N. Mathur, M.S., D.O.M.S. (Lond.), F.A.M.S., Principal, G.S.V.M. Medical College, Kanpur, for his permission to publish this report.

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News Briefs . . .

North Dakota

DR. LOWELL A. GESS, former medical missionary in Africa, joined the staff of the Quain and Ramstad Clinic in Bismarck as a specialist in ophthalmology.

* * * *

DR. LLOYD S. RALSTON, professor of medicine at the University of North Dakota and chief of medicine at the university's Rehabilitation Hospitals, joined the staff of the Grand Forks Clinic as a specialist and consultant in internal medicine.

* * * *

DISCUSSIONS on breathing problems of obese patients, care of the diabetic, hemolytic anemias, and treatment of depression in rural patients highlighted the 21st annual North Dakota meeting of the American College of Physicians at the University of North Dakota. Special guest was Dr. Carl V. Moore, chairman of the department of medicine at Washington University, St. Louis, Mo. Presiding at scientific sessions were Drs. Thomas E. Pederson, Jamestown, and Keith G. Foster, Dickinson. Dr. Robert M. Fawcett, Devils Lake, presided at the banquet. Other North Dakota speakers were Drs. Eric Barth, Donald M. Barnard, and John M. McMahon, all of Fargo; John A. Lambie, B. E. McLaughlin, and Russell H. Wilson, all of Grand Forks; Paul Klosterman, Bismarck; and Victor Kaljot, Minot. Drs. Harold W. Evans and Robert C. Painter, both of Grand Forks, were chairman of the program committee and in charge of general arrangements, respectively.

* * * *

DR. NORMAN KESTER, formerly of Manitoba, joined the staff of the Rolette Clinic.

* * * *

DR. CHARLES T. EGINTON, formerly a clinical assistant professor of surgery at the University of Minnesota, was named acting chief of the surgical service at the Fargo Veterans Administration Center, succeeding Dr. U. S. Anderson, who was transferred to the Minneapolis Veterans Hospital.

* * * *

DR. HECTOR GALLEGO, a native of Colombia, joined the staff of the Grand Forks Clinic as a specialist in skin diseases.

* * * *

DR. EUGENE BRASSEUR, previously associated with the DePuy-Sorkness Clinic of Jamestown as an ophthalmologist, joined the staff of the Professional Building.

* * * *

DR. JOHN C. LUND, formerly of Williston, completed the orientation course for officers of the U.S. Air Force Medical Service at Sheppard Air Force Base, Texas, and was assigned to Ellington Air Force Base, Texas, for duty with the Continental Air Command.

DR. MARK THOMAN, formerly with the U.S. Public Health Service in New Mexico, joined the department of pediatrics of the Quain and Ramstad Clinic in Bismarck.

Minnesota

DR. TED MYERS was elected president of the Freeborn County unit of the American Cancer Society.

* * * *

DR. ROLAND LARTER, a native of McKinley Township, opened an office at the Karlstad Clinic.

* * * *

DR. ROBERT PIERCE moved his practice from Mountain Lake to Heron Lake.

* * * *

EARLY RETIREMENTS for health reasons were announced by Drs. Jan Tilliseh, 58, and Maurice Stauffer, 52, both consultants in internal medicine at the Mayo Clinic, Rochester. A specialist in aviation medicine, Dr. Tilliseh has been medical director of Northwest Airlines since 1940.

* * * *

DR. DALE L. EICHELBERGER of Willmar was elected to active membership in the American Academy of General Practice.

DR. W. G. NUESSLE, who retired after nearly fifty years of practice in Springfield, donated his office fixtures and furniture for auction, with the proceeds going to the local community hospital.

* * * *

DR. HENRY BUCHWALD, assistant professor of surgery at the University of Minnesota, was awarded the $1500 Samuel D. Gross Prize in Surgery by the Philadelphia Academy of Surgery for his study of cholesterol metabolism and arteriosclerosis. He is the second University of Minnesota professor to have won the prize in the fifteen times it has been awarded since 1895.

* * * *

DR. ROBERT G. HANKERSON left his practice in Minnesota Lake to join the medical staff of the Mankato State College dispensary.

* * * *

DR. WINSTON R. MILLER, medical director of the St. Paul Medical Center, was appointed program director of the Northlands Regional Medical Program, a cooperative effort of 8 health and medical services to bring better care to heart disease, stroke, and cancer patients in Minnesota and surrounding areas.

* * * *

A TWO-DAY COURSE in "New Concepts in Problems of Completed Stroke" will be held Jan. 19-20 at the Kenny Rehabilitation Institute, Minneapolis, under the auspices of the American Rehabilitation Foundation.

(Continued on page 12A)

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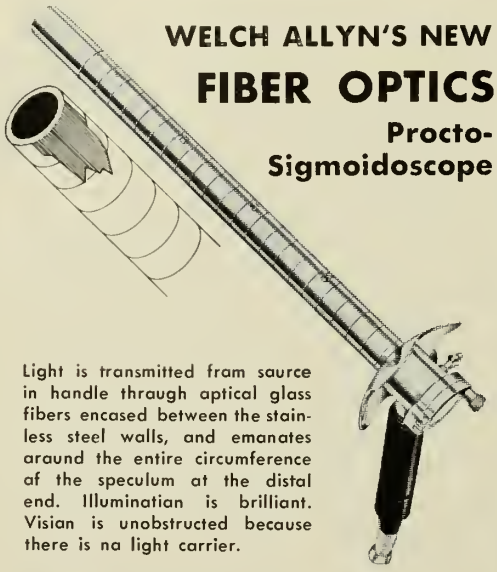
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NEWS BRIEFS

(Continued from page 11A)

DR. ROY RITTS, JR., will replace Dr. Lyle Weed as head of the Mayo Clinic section of microbiology on Jan. 1, 1968, it was announced. Dr. Weed will become a senior consultant. Dr. Ritts, 38, has resigned as director of medical research of the American Medical Association (AMA) and as director of the AMA-Education and Research Foundation Institute for Biomedical Research to accept the clinic position.

* * * *

DR. B. H. JOHNSTON left the Ivanhoe Clinic for service with the armed forces.

* * * *

DR. DOMINADOR B. SORIANO, a native of the Philippine Islands, joined the medical staff at the Cuyuna Range Clinic in Crosby.

* * * *

DR. DUDLEY WILKINSON, formerly of Silver Bay, joined the Peterson Clinic in Mora.

* * * *

DR. ROBERT A. GOOD and associates at the University of Minnesota received the first payment of $43,494 of a $78,328 grant from the National March of Dimes Foundation for birth defects research.

* * * *

DR. PAUL GERSTER, formerly of Farmington, joined the staff of Mork Clinic, Anoka, after ten years service with the U. S. Army Medical Corps.

* * * *

DR. ROGER J. BENJAMIN, a native of Pipestone, began practice in Owatonna.

* * * *

DR. JURGEN J. MOLLER, a University of Minnesota resident in anesthesiology, left for Vietnam as the seventh state physician to participate in the Volunteer Physicians for Vietnam program administered by the American Medical Association.

* * * *

DR. ERNST SIMONSON, chairman of the clinical section of Mount Sinai Hospital, Minneapolis, was honored at a banquet in conjunction with an international conference on his specialty, exercise cardiography, held at the Minneapolis Veterans Hospital.

* * * *

DR. MARIO M. FISCHER, retired St. Louis County director of public health, was cited by the Duluth Unit of the American Cancer Society for his "long and outstanding service to the cause of cancer control." Later in the same week, he was presented with a life membership in the Tuberculosis and Health Association of Northeast Minnesota.

SPEAKERS on the pathogenesis and treatment of rheumatoid arthritis at the Ninth Annual Medical Symposium at Methodist Hospital, Minneapolis, included Dr. Morris Ziff of the University of Texas, Dallas; Dr. Howard F. Polley of the Mayo Clinic, Rochester; Dr. Adrian Flatt of the University of Iowa, Iowa City; Dr. Paul J. Bilka of the University of Minnesota; Dr. Charley J. Smyth of the University of Colorado, Denver; and Dr. Donald F. Hill, president of the American Rheumatism Association, Tucson, Ariz.

* * * *

DR. JOHN IVINS, a consultant in orthopedic surgery at the Mayo Clinic, Rochester, was appointed to a 20-member Emergency Health Preparedness Advisory Committee by U.S. Surgeon General William Stewart. The Committee, which replaces a Civil Defense organization and the Surgeon General's Professional Advisory Committee for Emergency Health Preparedness, will assist in planning for health services and casualty care in case of natural disasters, enemy attack, or major emergencies in metropolitan areas.

* * * *

RESEARCH GRANTS totaling $11,400 were awarded by the American Cancer Society to 3 Mayo Clinic physicians, as follows: Dr. Richard Winkelmann, dermatologist, $6,000; Dr. Roy Shorter, surgical pathologist, $3,000; and Dr. Jurgen Ludwig, anatomical pathologist, $2,400.

* * * *

DR. H. D. MORI of Grand Meadow was honored with a plaque and an office stereo system for his community service.

* * * *

DR. HERBERT BOYSEN was appointed medical director of the Luther Memorial Home, Madelia.

* * * *

NEW OFFICERS of the recently reorganized Multi-County Health Services, Inc., a nonprofit corporation providing part-time home health services to the ill and disabled residents of Mahnomen, Becker, and Hubbard counties, include Dr. V. A. Watson, Detroit Lakes, chairman; Dr. W. R. Koons, Mahnomen, vice-chairman; and Dr. Clinton Rhodes, Park Rapids, director.

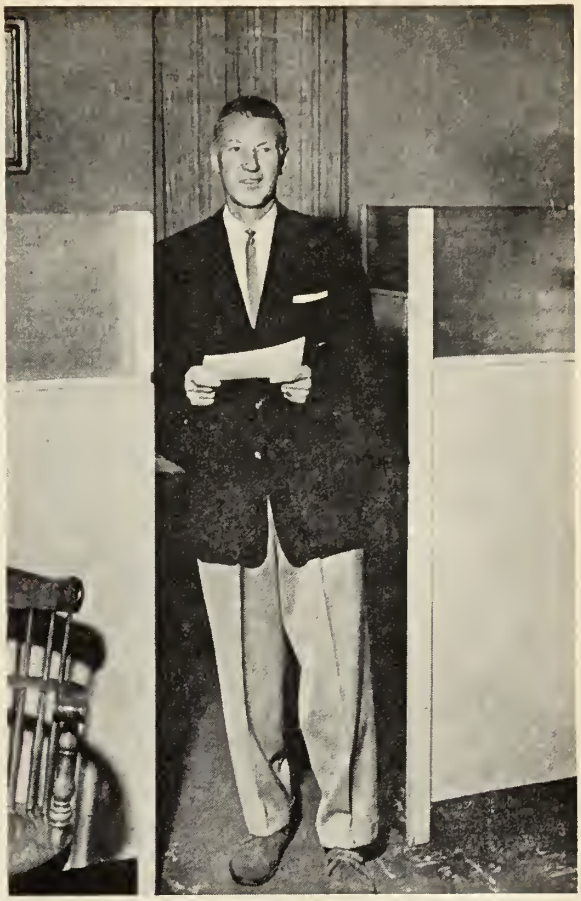
* * * *

DR. EUGENE RONDEAU left the Two Harbors Community Health Center Clinic and Lake View Memorial Hospital staff after ten years service for an assignment as general physician at the state mental hospital in Sedro Woolley, Wash.

South Dakota

DR. DAVID ROUSSEAU, a graduate of Marquette University, joined his father, Dr. M. C. Rousseau, in general practice at the Brown Clinic in Watertown.

(Continued on page 14A)



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NEWS BRIEFS

(Continued from page 13A)

DR. HENRY DAVIDSON, formerly of Sioux Falls, joined the psychiatric staff of Fort Meade Veterans Hospital.

* * * *

DR. PAUL ISAAK, formerly of Parkston, was named Alaska Physician of the Year by the Alaska State Medical Association. Dr. Issaak and a partner serve an area with a population of about 8,000 within a radius of 50 miles from Soldatna on Kenai Peninsula. They fly their own planes to attend patients and usually use a nearby road as a runway. They also fly patients to Seward or Anchorage hospitals, 100 miles away.

* * * *

DR. CHRIS J. MOLLER of the Dell Rapids Clinic, a veteran of the Korean War, left for Vietnam to participate in the Volunteer Physicians for Vietnam program administered by the American Medical Association.

* * * *

DR. WALTER KITZLER, formerly of Ipswich, took over the Gettysburg office and practice of Dr. E. W. Urbanyi, who has joined the staff of the Veterans Administration Hospital at St. Cloud, Minn.

* * * *

DR. JOHN FREEMAN, a native of Texas, joined the Hand County Clinic, in association with Drs. John G. Hagin and James H. DeGeest, at Miller.

* * * *

DR. W. A. STEPHENS, formerly of Custer, opened an office in Murdo.

Deaths . . .

DR. NORMAN W. ANDERSON, 63, a native of Minneapolis, Minn., died in Topeka, Kan., Sept. 11. A thirty-year veteran of the U.S. Army Medical Corps, he was director of the medical care division of the Kansas Department of Health at the time of his death.

* * * *

DR. OLIVER PORTER, 90, formerly of Atwater and Willmar, Minn., died in Minneapolis, Sept. 6. He had practiced in Sisseton, S.D., for twenty years before coming to Minnesota after World War II.

* * * *

DR. WILLIAM C. FERGUSON, 57, Walnut Grove, Minn., died Sept. 5. A native of New Mexico, he had practiced in Walnut Grove for thirty years.

* * * *

DR. J. A. COSGRIFF, SR., 75, Olivia, Minn., died Sept. 4. He had practiced in Olivia for thirty-eight years and was national president of the Association of American Physicians and Surgeons. He was past president of the Minnesota Academy of General Practice and the Minnesota Heart Association.



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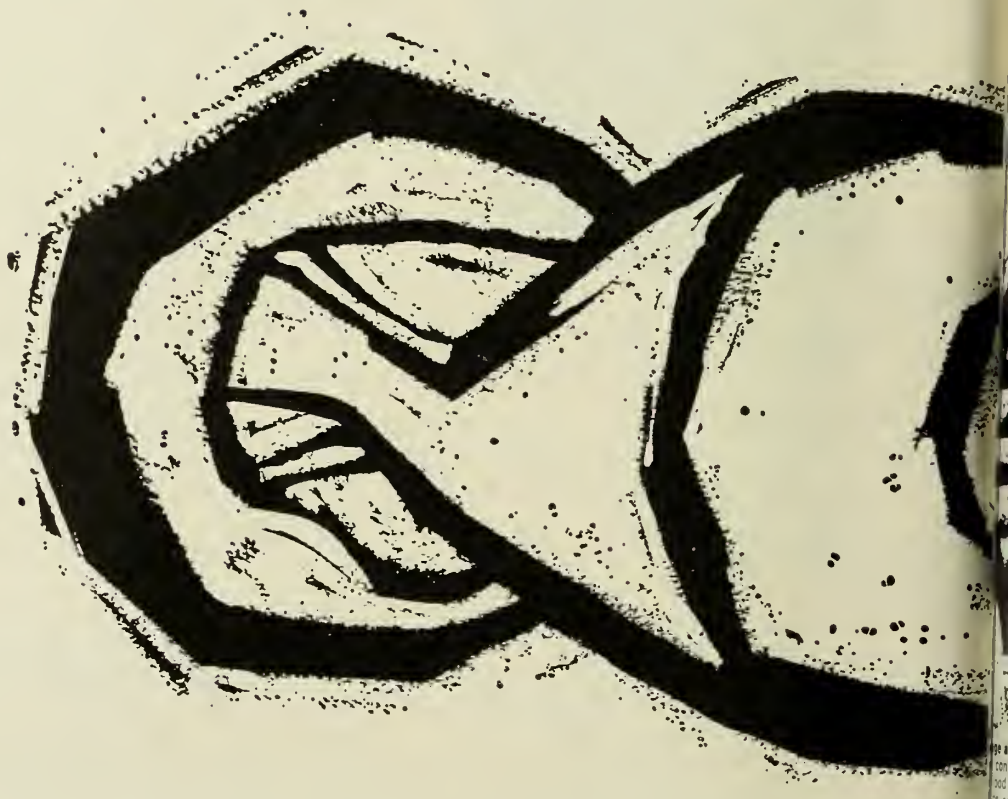
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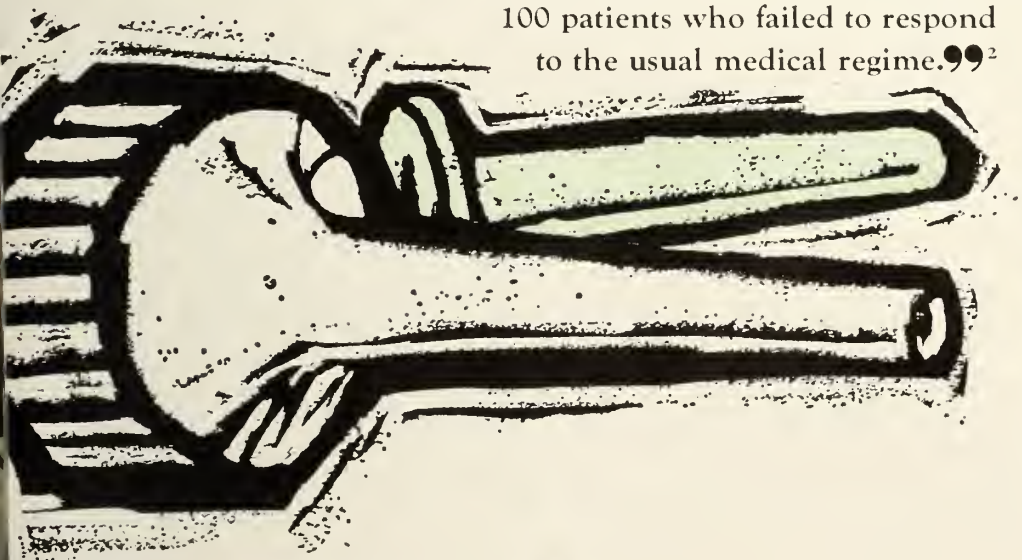
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2. Sherman, L. F., Tenner, R. J. and Schottler, J. L.: Treatment of ulcerative colitis with hydrocortisone alcohol retention enemas: A topical and systemic anti-inflammatory agent. Part II, *Dis. Colon & Rectum* 10:43, January-February 1967.



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CONTENTS

Complete Heart Block Associated with Pregnancy and Treated with an Internal Pacemaker 461
S. A. Berestka, M.D., and W. N. Spellacy, M.D.

Oral Roentgenographic Changes in Wilson's Disease 464
Richard G. Ogle, D.D.S., Joseph A. Gibilisco, D.D.S., Norman P. Goldstein, M.D., and Raymond V. Randall, M.D.

Copper and Calcium Metabolism in the Rat: Relationship to the Pineal Gland 467
R. E. Wiederanders, M.D., and G. W. Evans, B.S.

Eosinophilic Peritonitis: Report of Two Cases 473
Carl E. Hunt, M.D., Theodore C. Papermaster, M.D., Edward N. Nelson, M.D., and William Krivit, M.D.

Series on Urology for the General Practitioner
Carcinoma of the Prostate: A Review: Part I. Incidence, Etiology, Pathology, and Diagnosis 477
Clyde E. Blackard, M.D.

Henry L. Williams, Jr., M.D. 485
Jerome A. Hilger, M.D.

Index to Volume 87 487

Letters to the Editor 497

News Briefs 10A

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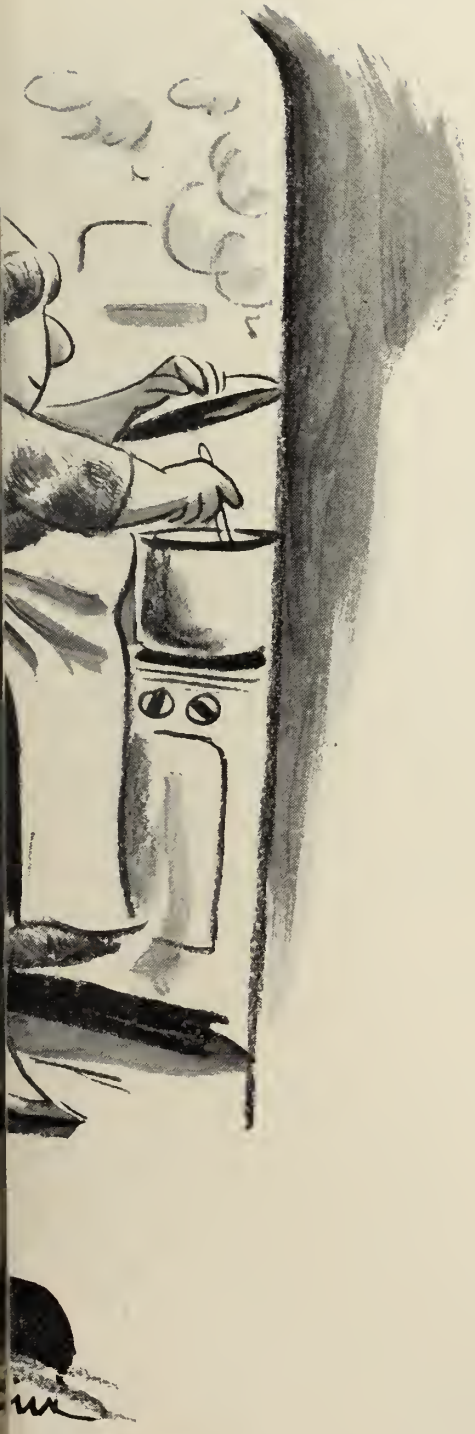
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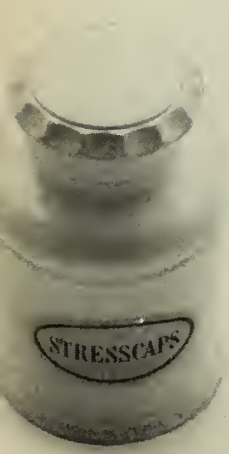
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Complete Heart Block Associated with Pregnancy and Treated with an Internal Pacemaker

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Minneapolis

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Miami

HEART BLOCK is a rare complication of pregnancy probably because 90% of the cases occur after the age of 50 years and because the problem is twice as common in males as in females.[1] The use of cardiac pacemakers during pregnancy is a new form of treatment. In the American literature there is only 1 report of a patient who had a complete heart block treated with an internal pacemaker and who subsequently became pregnant.[2] The purpose of this paper is to present a case of complete heart block diagnosed and treated during pregnancy with an internal cardiac pacemaker.

CASE HISTORY

A 20-year-old unmarried, primiparous was referred to the University of Minnesota Hospital on Sept. 2, 1964, because of a complete heart block. This was diagnosed elsewhere when the patient sustained a fractured right ankle July 5, 1964, while falling during an apparent Stokes–Adams attack. An electrocardiogram had confirmed the diagnosis with a heart rate of 20 beats per minute. Despite medical management she had 2 more Stokes–Adams attacks during the ensuing eight weeks. She was then referred for pacemaker implantation.

The admission history was significant in that the patient denied previous cardiac symptoms. She had played sports without difficulty and had had 3 other hospitalizations for minor surgery without a diagnosis of cardiac disease. The patient was pregnant with an expected date of confinement of Jan. 4, 1965. The physical examination showed a normal female with a regular pulse of 56 per minute and a blood pressure of 110/60. The second heart sound was split with the pulmonary component being louder than the aortic component. There was a grade II/VI systolic ejection murmur heard at the base and along the left sternal border. The lung exam was normal. The abdominal examination showed a pregnant uterus compatible with her dates.

On Sept. 12, 1964, she underwent cardiac catheterization studies. At rest the pulse rate was 60 to 62 beats per minute and with exercise it rose to 72 beats per minute. The rise in pulse rate with exercise suggested that this was a congenital type of heart block. At rest the cardiac output was 7.4 liters per minute and with exercise it rose to 10.9 liters per minute.

On Sept. 14, 1964, a fixed-rate internal pacemaker* was placed in the left ventricle and set at a rate of 76 beats per minute. Because the patient was pregnant the battery was implanted under her left breast (Fig. 1A and 1B). Postoperatively the patient's course was complicated by pneumonia and a sterile empyema.

On Oct. 6, 1964, a repeat cardiac catheretization was

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*This pacemaker is supplied by Medtronic, Inc., Minneapolis, Minnesota.

done. At rest the heart rate was 78 beats per minute with all beats arising from her pacemaker. The cardiac output was 6.7 liters per minute. With exercise the rate rose to 84 beats per minute because of escaped beats from a presumed nodal focus. The cardiac output with exercise was 10 liters per minute. Repeated electrocardiograms showed a right bundle branch block.

Although the patient was considered to be a functional class 1 to 2, she was kept in the hospital for the remainder of her pregnancy because of the lack of knowledge about this condition. Periodically, she would complain of pain under her left breast. There were no further syncopal attacks. Her vital capacity, venous pressure, and circulation time were monitored weekly and they all remained normal. Her total weight gain was 14 lb.

On Jan. 29, 1965, an intravenous pitocin induction of labor was accomplished. After a total labor of nine hours and twenty minutes she spontaneously delivered a normal 3,945 gm. female infant over a left mediolateral episiotomy using pudendal block anesthesia. During the labor and delivery her pulse was recorded between 78 to 80 beats per minute. Following delivery a uterine atony resulted in the blood loss of 650 ml. This responded to oxytocin stimulation. She was discharged home on the eighth postpartum day. The patient has been followed in cardiac clinic and has done well.

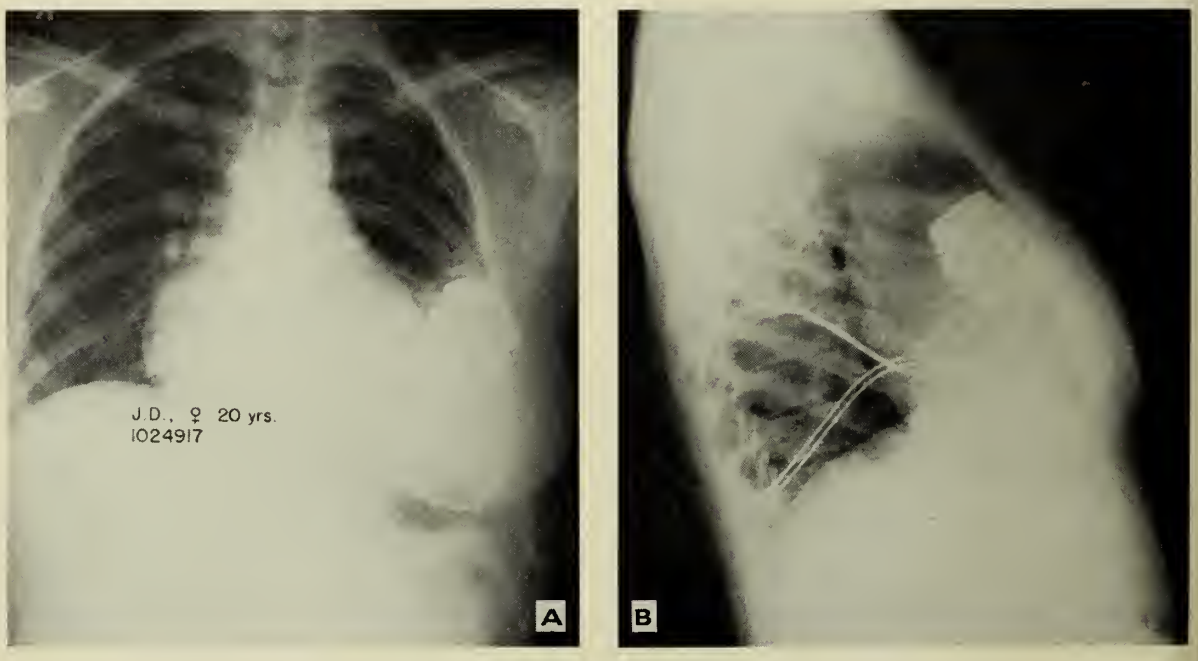
PATHOPHYSIOLOGY

In complete heart block no impulses get from the auricle to the ventricle. The ventricles thus escape and contract at their inherent rhythm which is usually slow, 30 to 40 per minute. Approximately one-half of the patients with congenital heart block have an associated interventricular septal defect. In acquired heart block the etiological factors include rheumatic fever, syphilis, diphtheria, coronary artery disease, or excess digitalis.[1]

The complications of the slow heart rate are Stokes–Adams attacks and congestive heart failure. In animal experiments congestive heart failure resulting from surgically induced heart block may be followed by spontaneous clinical improvement despite the persistence of the block and bradycardia.[3] This has also been found to occur in patients. The adaptation is accomplished by several mechanisms: (1) The heart develops a larger stroke volume, (2) there is an increase in peripheral oxygen extraction, and (3) there is a decrease in the basal metabolic rate.[3] In some patients the ventricular rate is fixed, with only minimal rate changes between rest and work. However, a rather moderate increase in pulse rate with exercise is used to differentiate congenital from acquired heart block.[4] Nevertheless, the cardiac output remains below normal both at rest and during work. There are occasionally other beats which get through to cause a variation in the rate.

SYMPTOMS AND DIAGNOSIS

When complete heart block has become established patients may be without symptoms at rest, but may experience dyspnea and dizziness on exertion. Other symptoms which sometimes occur are palpitation, fainting, and convulsions. The diagnosis is dependent upon a clinical sus-



Chest X-ray during pregnancy showing cardiac pacemaker implanted under left breast and on myocardium

picion when one detects bradycardia. An electrocardiogram confirms the diagnosis.

MANAGEMENT

Many forms of medical treatment for heart block have been used including barium chloride, sedation, thiazides, cortisone, ephedrine, atropine, isoproterenol (Isuprel®), and intravenous hypertonic sodium lactate.[5–7] In 1952 Zoll described the successful treatment of a patient with repetitive electric shocks to the chest wall.[8] With the development of artificial pacemakers a direct electrical stimulation of the cardiac muscle became possible. A ventricular contraction results from the stimulus unless it arrives during the refractory period from a previous contraction. This form of therapy has greatly reduced the morbidity and mortality of this disease.[4] The pacemaker is implanted subcutaneously and encased in a material which is free of tissue irritation. Impulses then are delivered directly to the heart electrode by wire. The pacemakers are generally set at a fixed rate of 60 to 70 beats per minute. Recent work has suggested that a pacemaker with the frequency dependent upon atrial activity might be preferable.[8] The batteries last from three to five years and can be replaced using local anesthesia. Recently it has been found that by means of chest X-ray examination every six months, it is possible to predict battery failures many months in advance and thus give time for battery replacement.[9]

HEART BLOCK AND PREGNANCY

Nanta described the first case of heart block in a pregnant woman in 1914.[10] Since then several individual cases have been reported. Mowbray's review of the literature up to 1948 found only 35 but Stokes–Adams attacks were infrequent and cardiac failure symptoms were minimal. Vaginal delivery presented no problems and the postpartum course was uneventful.[10] In 1951 Epstein and Altman reviewed 45 cases and concluded that the maternal prognosis depended upon the cardiac reserve at the first visit. The overall maternal mortality was 13.4% and the acquired type of block did poorer than the congenital type.[11] Mendelson reviewed the literature and added 5 cases of his own in 1956. He concluded that none of the reported fetal deaths were directly related to the heart block.[12]

Before pacemaker therapy an occasional patient had to be treated by therapeutic abortion.[13] This is not generally advised today.[6]

The use of pacemakers in the treatment of pregnant women is a new concept. Shouse and Acker have reported a patient who had a cardiac pacemaker implanted before pregnancy. The patient progressed normally through the pregnancy with a pacemaker set at 72 beats per minute. The only complication was a sudden increase in the heart rate eight hours after delivery.[2] The local irritation of the pacemaker seems to be one complication for the pregnant woman. Büchner and associates reported a case where the pacemaker was implanted into the left rectus sheath during pregnancy and pain occurred during the fourth month.[14] The present case had irritation of her breast by the implanted pacemaker as the breast enlarged. A perfect implantation site for the young female is not known.

Although the catherization studies performed on this patient failed to indicate any improvement in the cardiac output with the pacemaker in place, the lack of any further syncopal attacks indicated its usefulness. One can easily speculate as to the fetal dangers resulting from hypotension.

CONCLUSION

Heart block is a rare complication of pregnancy. The morbidity seems to be higher for the acquired lesions than for the congenital ones. Generally, the symptoms are mild during pregnancy. Management can usually be successfully carried out by combined medical and surgical (pacemaker) approach with excellent results. A case history is presented.

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Oral Roentgenographic Changes in Wilson's Disease

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RAYMOND V. RANDALL, M.D.

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A STUDY WAS MADE of 16 sets of full-mouth intraoral roentgenograms of 11 patients with Wilson's disease. Of the patients surveyed, only 1 showed no changes; the remaining patients showed at least 1 abnormal roentgenographic finding. The principal changes seen were (1) demineralization of bone (local or generalized decreased density), (2) alteration of trabecular pattern, and (3) alteration of landmarks and loss of alveolar structure. Hypercalciuria was noted in 9 of these 11 patients. The cause of the decreased bone density seen in these patients with Wilson's disease is not known.

Wilson's disease (hepatolenticular degeneration) is a rare, inherited (autosomal recessive) disease associated with an abnormality of copper metabolism. It is characterized pathologically by a coarsely nodular cirrhosis of the liver and a progressive degeneration of the central nervous system, particularly the lenticular nuclei. In view of the finding of hypercalciuria in some patients,[1–7] it is interesting that another abnormality of calcium metabolism has been found, namely, demineralization of the skeleton.[5,8] It is not clear what relationship there is, if any, between hypercalciuria and skeletal demineralization.

It occurred to us that dental disturbances might be another manifestation of these abnormalities in calcium metabolism. A survey of the literature revealed only a single reference to this topic. Morgan and co-workers[5] described 1 patient as having "idiopathic apical absorption" of the maxillary central incisors. They made no mention of associated osseous abnormalities in the jaws.

This paper describes the dental abnormalities found on roentgenographic examination of 11 patients with Wilson's disease.

MATERIALS AND METHODS

The case histories of these patients seen at the Mayo Clinic in the last ten years were reviewed. Among them, 11 had had 1 or more complete sets of intraoral dental roentgenographic surveys, totaling 16 such sets. These films were evaluated, independently initially and then jointly, by the investigators for bone and dental changes in 13 different categories.

Demineralization was judged on the basis of apparent loss of bone density and was graded arbitrarily on a scale of 0 to 3 (ascending values corresponding to increasing loss of bone density). Alteration of trabecular pattern was considered demonstrable when the quality and number of trabeculations were decreased (graded on an arbitrary scale of 0 to 3). Changes of anatomic landmarks were quantified on the basis of alteration of 3 major landmarks: the nasal aperture, the cortical outline of the antra, and the mandibular canal. The number of these structures altered became a grade on a scale of 0 to 3.

For comparison, 40 sets of full-mouth intraoral dental roentgenograms were examined. These were picked randomly from the files of the Mayo Clinic and represented 21 males and 19 females ranging in age from 11 to 83 years. The recording of abnormalities in these roentgenograms was limited to demineralization and trabecular change; grading was by the same scales used to evaluate the study cases. This group included adolescents with mixed dentitions and aged patients, who normally display reduced bone density in the jaws. To maintain consistency, grading was not altered to account for these normal variants in either the patients with Wilson's disease or the control patients.

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The urinary excretion of calcium was measured, during 3 twenty-four-hour periods, in each of the 11 patients while they were receiving a low-calcium diet. We consider a twenty-four-hour urinary calcium excretion in excess of 170 mg. under these circumstances to be hypercalciuria. The data given in the table represent the average of the values.

RESULTS

Among the 40 control roentgenograms, 17 were grade 0 (completely normal) for bone density and trabeculation, 21 were grade 1 (mild involvement) loss of bone density or alteration of trabecular pattern, and 2 were grade 2. None of these roentgenograms were considered to show severe involvement.

No congenital anomalies or abnormalities of tooth eruption or tooth maturation were found in any patient with Wilson's disease. Demineralization of maxillary or mandibular bone or both and alteration of trabecular pattern were the most common abnormal features (see table), and there seemed to be a correlation between these findings and clinical status. Also, these 2 changes were more frequent among the patients with Wilson's disease than among the comparison subjects.

There appeared to be no severe involvement of the lamina dura in any case. Pulp stones, from 1 to 3 per arch, were seen in 6 of the 9 dentulous films. Periodontal disease was noted in 5 of the dentulous films. One film displayed pulpal obliteration and 2 showed some abnormal pulp size. It was also thought that 2 patients displayed alteration of the periodontal membrane space. All patients in this series except 2 (cases 4 and 5) had hypercalciuria, and 8 with hypercalciuria displayed demineralization. One patient with normal urinary calcium values had severe demineralization of bone on the intraoral roentgenogram (Case 4) and, conversely, 1 patient with hypercalciuria did not demonstrate demineralization (Case 2).

One case (Case 4) was of particular interest, that of a 40-year-old female seen first in July

FINDINGS IN 11 PATIENTS WITH WILSON'S DISEASE

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*
Age (year) and sex	16M	20M	39M	40F	21F	32M	26F	33M	24F	17M	27M
Duration of illness (year)	½	1	2	8	4	7	10	2	1	7	4
Urinary Ca, mean of three days (mg./24 hours)*	357	246	200	124	122	239	288	347	179	362	435
Demineralization	2	0	3,2†	3,3,2	0	1	1	2,2,1	1	3	2
Trabecular pattern	2	1	2,1	3,3,2	0	1	1	1,1,2	1	2	2
Anatomic landmarks	1	0	0,0	2, . . . ,1	0	0	0	1,1,2	0	0	0
Periodontal disease	0	0	3,0	Edent.	0	Edent.	Edent.	1,1,1	2	0	0
Calcification of pulp	1	1	Edent.	Edent.	0	Edent.	Edent.	1,1,1	1	0	0
Lamina dura	0	1	Edent.	Edent.	0	Edent.	Edent.	0,0,0	1	0	0
Periodontal membrane	0	1	Edent.	Edent.	0	Edent.	Edent.	0,0,0	2	0	0
Pulp obliteration	0	1	Edent.	Edent.	0	Edent.	Edent.	0,0,0	0	0	0
Resorption of alveolus	0	0	Edent.	Edent.	0	Edent.	2	1,1,1	2	0	0
Size of pulp	1	0	Edent.	Edent.	0	Edent.	Edent.	0,0,0	0	1	0

*Values greater than 170 mg. per day represent hypercalciuria.

†Multiple values are grades in chronologic order when more than one set of roentgenograms were available.

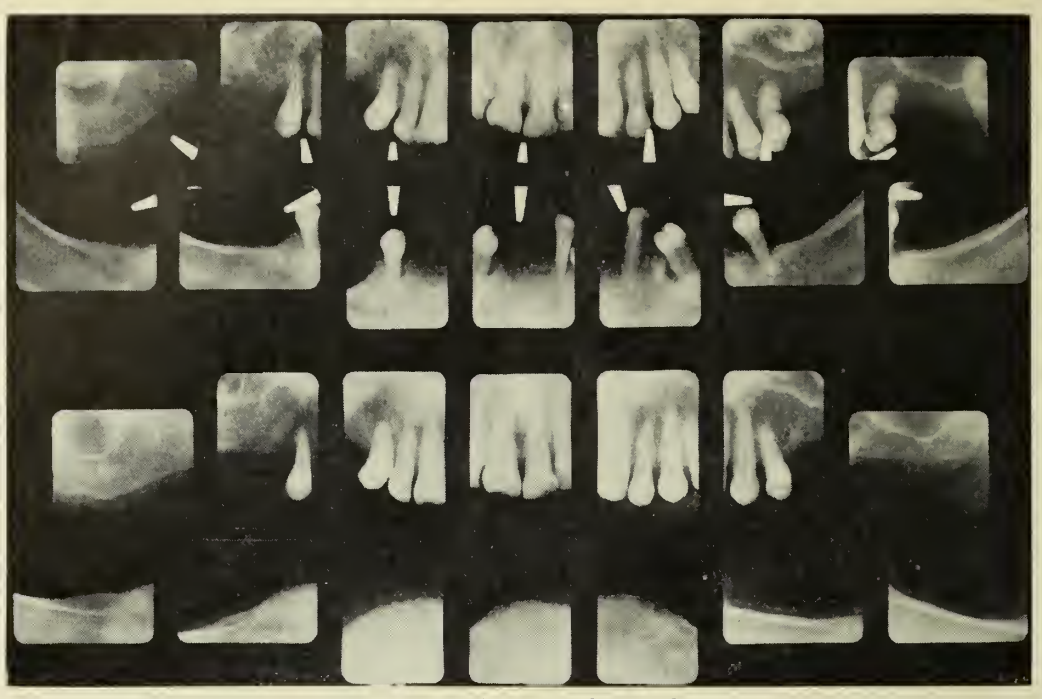


Fig. 1. (Case 4). Intraoral roentgenograms of 40-year-old female. *Top set.* Taken in 1963, these displayed severe loss of bone density, loss of trabecular pattern, and some obliteration of landmarks. These changes are best seen in molar-bearing areas of mandible and maxilla. *Bottom set.* Taken thirty-five months later, these show definite remineralization. Note return of trabecular pattern in posterior mandibular region and apparent increase in density in left posterior maxillary and anterior mandibular regions (Fig. 2). There is also a more distinct cortical outline in the posterior part of mandible.

1962. At that time she had had Wilson's disease for eight years. Her symptoms had been progressive and, at the initial visit, she had been bedridden for six months. A course of treatment was begun including DL-penicillamine (later, D-penicillamine) and a diet low in copper. Her clinical response was excellent, and currently the patient is leading a relatively normal life. It is interesting that her dental roentgenograms taken in 1963, 1964, and 1965 show signs of definite remineralization (Figs. 1 and 2).

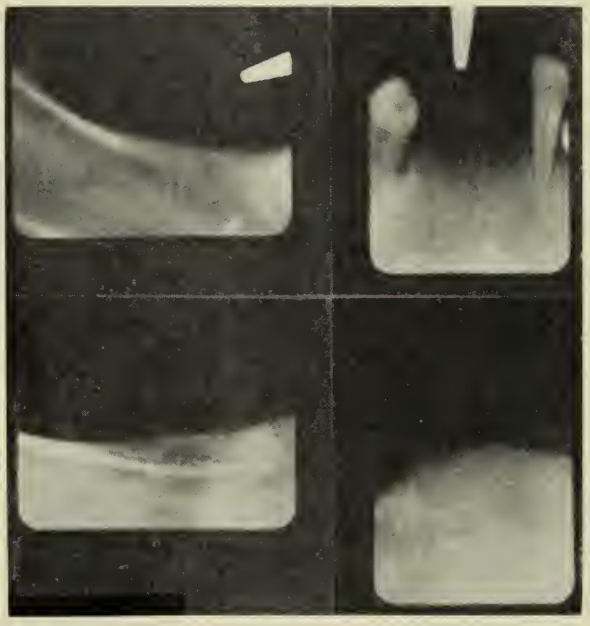


Fig. 2. (Case 4). Higher magnification of selected views from Fig. 1. *Upper,* 1963; *lower,* thirty-five months later.

DISCUSSION

It is interesting that there were no apparent alterations in the growth or development of the teeth or jaws in any of the patients with Wilson's disease. Also, it is interesting that of the 11 patients, only 1 had no dental abnormalities and this patient was 1 of the 2 without hypercalciuria. However, since none of the 11 patients had evidence of demineralization on roentgenographic surveys of the skeleton, it is not clear what relationship, if any, exists between the finding of demineralization in dental roentgenograms and the presence of hypercalciuria. It should be noted that, although decreased bone density in the jaws may be seen in conditions such as hyperparathyroidism, osteomalacia, gen-

(Continued on page 472)

Copper and Calcium Metabolism in the Rat

Relationship to the Pineal Gland

R. E. WIEDERANDERS, M.D., and G. W. EVANS, B.S.

Williston

Only in recent years has pineal physiology been rescued from the midst of mysticism to become a respectable study of science. Phylogenetically, the pineal is believed to be a remnant of the "third eye" of the lizard. There, it serves a function of sight in conjunction with the lateral eyes, oddly enough being sensitive to ultraviolet and visible light but not to infrared radiation.[1] In the mammal, it is no longer adapted to receive light but functions, instead, as an endocrine gland throughout life.[2]

Since it is derived from the diencephalon, it would seem that the pineal would be innervated via nerves from the habenula, but this is not the case. These nerves simply enter the gland and loop back into the habenula.[1] It is the sympathetic nervous system, extending bilaterally from the superior cervical ganglion, that sweeps over the cerebral convexities to thicken into the nervi conarii and enter the tip of the pineal, there to attach intimately to the pineal parenchymal cells. They attach, and presumably transmit, their impulses via serotonin in the rat, mouse, guinea pig, dog, hamster, pig, cow, and sheep and noradrenaline in the cat and rabbit.[1] The serotonin occurs in two pools—one in the sympathetic nerves and the other in the pinealocytes.[1]

Thus innervated, the gland is linked via the lateral eyes to the circadian cycle.[3] The rat, being most studied, is the best example to illustrate this process. Light stimulates 5-hydroxytryptophan decarboxylase activity to produce serotonin,[4] whose midday level averages about 90 ng. per pineal. With the onset of darkness, serotonin is depleted at the rate of 25 ng. per hour to a midnight low of 10 ng. per gland.[5] This decrease is partly accounted for by conversion of serotonin to N-acetylserotonin and, secondarily, O-methylation of N-acetylserotonin to melatonin via an enzyme peculiar to the pineal gland—hydroxyindole-o-methyl transferase (HIOMT).[6] Thus, melatonin is the peculiar product of this gland.[7] Serotonin production continues to build up and decline according to the circadian cycle in blinded rats and those kept in constant darkness, but this rhythm is abolished by constant light.[8] Darkness stimulates the activity of HIOMT and, thus, the production of melatonin, while light suppresses it.[9] This is the situation in the rat, a nocturnal animal, but in the hen, a diurnal animal, it is exactly reversed,[10] with serotonin produced at night and melatonin, during the day.

For some forty years it has been known that pineal extracts would cause lightening of skin color when injected into frogs, toads, and fishes. Recently, melatonin has been shown to be responsible for this action by causing the melanin granules in the melanocyte to cluster near its nucleus.[11] Melanocyte-stimulating hormone from the pituitary disperses these granules and leads to skin darkening in amphibians but not in mammals, whose pineals produce the greatest quantity of melatonin.[12] Other actions of melatonin are suppression of the estrous and prolongation of the diestrous phase of the sex cycle, decrease in ovarian[13,14] and male genitalia size,[1] suppression of thyroid function,[15] and possible depression of aldosterone release via ubiquinone,[16] although this is controversial.[1]

Recent studies suggest the possibility that the pineal may influence neoplastic growth. While the presence of calcification in the gland is not related to neoplasia,[17] pinealectomized animals proved more susceptible to Walker 256 carcinoma and died more quickly.[18] Human pineal glands are found to be considerably enlarged in patients who have died a cancerous death compared with patients who have died a noncancerous death.[19]

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This work appears quite solid and reproducible, but other areas of investigation have produced uncertain results. In fact, a most excellent recent study in the chick shows no function at all from the gland.[20] Three of these controversial areas have attracted our attention: calcium, glucose, and copper metabolism.

The parathyroid glands are reported to hypertrophy following pinealectomy in the rabbit but not in the dog or chick, while in the rat the principle parathyroid cells are increased in size by epiphysormone administration. This substance decreased the tetany response in thyroparathyroidectomized dogs.[13] No change in the blood calcium occurs in the pinealectomixed fowl, and the results of pineal extract administrations are inconclusive.[13] Pineal gliosis and atrophy follow a calcium-free diet in the fowl. The rat pineal hypertrophies after rib fracture, but the rate of callus formation is unchanged.[13] Thus, it appears that the pineal stimulates the parathyroids in the rat. Three experiments were planned around this: [1] the plasma calcium level in normal and pinealectomized rats as most directly mirroring the state of parathyroid function, [2] the rate of epiphyseal closure which is dependent on parathyroid activity and many other factors, as well, and [3] the production of calciphylaxis which entails massive movement of calcium in the body. This is the formation of calcified plaques in all layers of the skin occurring in animals sensitized by a systemic calcifying factor and subsequently challenged by injection of such substances as egg almumin.

The fasting blood sugar (FBS) has been shown to increase significantly in the rabbit but not in the fowl after pinealectomy, while pineal extracts gave widely varying effects.[13] It seemed worthwhile to look for this effect in the rat.

The third area, copper metabolism, doesn't seem to have been linked to this gland previously. But if one considers that the blood copper level varies with the circadian cycle,[21] then it seems reasonable to look for pineal control of copper. In the human being, an increase of as much as 15% occurs in the blood copper level during the hours of activity.[21] Determinations of blood copper, plasma copper, and ceruloplasmin levels in the adult rat and tissue copper in the young rat have been made to test this hypothesis.[22]

METHOD

Wistar rats were used throughout the study. They were purchased at 30 days of age and, from that time on, kept strictly on Purina Lab Chow containing 15.7 μg. of copper per gram and tap water with 23.6 μg. of copper per liter, as analyzed in this laboratory.[22] Pinealectomized rats and normal controls were used. The gland was removed at 30 to 32 days of age unless otherwise stated; as all the results are negative, sham-operated animals were used in only two experiments. Pinealectomy was done according to Burklund's method,[23] in which the animals are anesthetized with intraperitoneal pentobarbital anesthesia. Through a midline scalp incision, a 1-cm. cut along the sagital sinus is made, beginning at its confluence with the lambdoidal suture and extending along the lambdoidal suture for 0.5 cm. The ends of these cuts are joined by parallel incisions and the bone flap, removed. The right cerebral hemisphere is depressed with a sponge, and a jeweler's forceps is used to reach under the sinus confluence, grasp the gland by its stalk, and remove it. It is recognized as globular and tends to be bilobed and translucent. A small piece of hemostatic sponge is inserted in the pineal bed and the skin sutured. This technique is most adaptable to the newborn rat where the cuts along the sutures are made but the bone flap is not cut out. It is simply depressed with the forceps and the gland removed. Hemostatic sponge is not necessary.

Blood was obtained by transsternal cardiac puncture, between 5 and 8 P.M. Plasma calcium was measured by standard methods on the flame photometer. Selye's[24] method of inducing calciphylaxis was followed to the letter: Each animal was weighed and given 1 mg. of dihydrotachysterol in corn oil per 100 gm. of body weight by stomach tube. Two days later, 10 cc. of egg white were injected at multiple points into the flanks, with care taken to miss the legs and thorax. Epiphyseal closure time was judged by X-ray. Each rat was given 3 mg. of pentobarbital per 100 gm. of body weight intraperitoneally and then laid on its side on cardboard-protected industrial film. This was done weekly up to the second month of life and at monthly intervals thereafter.

Blood sugar was done by usual laboratory methods and is reported as true blood sugar. Total blood copper and ceruloplasmin are measured, respectively, by the techniques of Gubler and co-workers and Houchin, and in each case their technique was followed exactly.[25,26] Ceruloplasmin is not measured directly by this method but, rather, its oxidase activity is assessed, which is proportional to its concentration. Oxidase activity is converted to ceruloplasmin by multiplying optical density by 157, the con-

stant we have determined for the rat. Plasma and tissue copper were determined by the wet-ashing method,[22] using nitric, perchloric, and sulfuric acids to digest the tissue. The spectrophotometer was used to read the blue color produced by copper in the presence of oxalyldihydrazide-acetaldehyde.

Some data are presented as the arithmetical mean with standard deviations and other data, as means and extremes. None of this data showed significant difference by standard tests.

RESULTS

Calciphylaxis was attempted in 35 pinealectomized rats and 36 normal controls when the rats weighed between 100 and 150 gm. An arbitrary scale was devised to measure the degree of calciphylaxis as follows: An area of 2 sq. cm. or smaller is called 1+, 2 sq. cm. to 4 sq. cm. is 2+, 4 sq. cm. to 6 sq. cm. is 3+, and anything over this is 4+ involvement. Four pinealectomized and 3 control rats failed to develop the characteristic calcified plaques. Within six days of the egg-white injection, 13 pinealectomized and 8 control rats were dead. By thirty days, the group had evened out because 2 more pinealectomized rats and 11 more controls had died. In each group, the maximum calciphylaxis response was seen from seventeen to twenty days and divides itself as follows: The pinealectomized group had two rats with 1+, three with 2+, one with 3+, and ten with 4+ reactions, whereas the control group had one with 1+, two with 2+, one with 3+, and ten with 4+ reactions. In each group, the area had sloughed and healed by six weeks. All animals developed permanent scars but some hair grew back over the affected sites.

Epiphyseal closure time is an uncertain determination at best and, as can be readily seen, there are wide variations. Two groups of animals were used. In the first, consisting of 19 control, 22 pinealectomized, and 4 blinded animals, the calcaneous epiphysis closed from 100 to 119 days and the glenoid epiphysis of the scapula closed from 91 to 126 days of age. All the vertebral bodies had fused in both groups between 111 and 280 days. The upper fibular epiphysis closed in both groups from 280 to 375 days. Obviously, there is no difference between the normal and pinealectomized groups here. The four blinded rats showed fusion of the glenoid scapular epiphysis and the calcaneous epiphysis at 88 days; however, the vertebral bodies fused between 120 and 150 days, which is in accordance with the other rats. A second group of 24 rats in 3 litters had pinealectomy in half of each litter and the sham operation in the other. Because it was thought that the pineal might produce an effect in the neonatal period which the first group missed, the operations were performed on the day of birth. All these animals were X-rayed at 90 days of age. Again, the state of epiphyseal closure was identical.

Blood studies were made when the animals reached 6 months of age. Average plasma calcium values on 56 normal and 51 pinealectomized rats showed a mean of 4.9 and 4.8 mEq. per liter, respectively. These values were too close to warrant calculating the standard deviations. Fasting blood sugar values were farther apart, with the average for 87 control animals being 68.6 ± 10.7 and for the 77 pinealectomized rats, 72.3 ± 10.3 mg. per 100 cc.

Total blood copper and ceruloplasmin determinations on 87 control rats gave results of 1.5 ± 0.8 μg. per cubic centimeter and ceruloplasmin level of 40.0 ± 10.5 mg. per 100 cc. Eighty-four pinealectomized animals had a mean blood copper of 1.5 ± 0.7 μg. per cubic centimeter and a ceruloplasmin level of 42.4 ± 12.3 mg. per 100 cc. Again, there is no difference. Plasma copper was determined by the wet-ashing method on 30 normal and 10 pinealectomized rats. Their respective means and extremes were 1.36 (1.09-1.76) and 1.44 (1.03-1.85) μg. per cubic centimeter. Again, no difference was found. The mean of all 40 animals is 1.38 μg. per cubic centimeter.

The last area of copper metabolism to be examined was the tissue deposition in the baby rat. Six litters, containing 55 animals, were divided in half on the day of birth. One-half had pinealectomy and the other half was sham operated. At autopsy, the four organs indicated on the graph were removed and weighed wet to the nearest milligram on a sartorius balance. Either the entire organ or a weighed aliquot was digested and analyzed for copper. The graph shows the number of animals in the litter on the day the litter was sacrified. The arithmetical means of the copper concentration as micrograms per gram are plotted and indicate clearly that the pinealectomized and sham-operated animals have the same concentrations of copper.

DISCUSSION

This approach to removing the pineal gland works well in the rat, where it sits up like a berry on its stalk, but we have been unable to use this method in the rabbit. By not unroofing the area, the venous sinus is opened on only one

side, and this readily stops bleeding by tamponade between the cerebral hemisphere and the skull. It is similar to the technique of Quay,[1] except we use pentobarbital (Nembutal®) instead of ether and the meninges are not cut. The forceps are simply pushed through them to the gland. Only a small piece of bone is removed, and little brain damage need be done. The whole operation requires less than three minutes. A theoretical disadvantage is that one may not remove all of the stalk; however, we have not seen evidence of its recurrence up to a year. Another drawback is that one cannot directly see the gland, but with experience, it may be readily removed and is easily recognized. The operative mortality is that of the anesthetic agent used. Our method is most useful in baby rats, where little brain damage is done and almost no blood is lost.

We chose three windows to look in on calcium metabolism, but all certainly failed to disclose any difference between the normal and pinealectomized rat. Because the blood calcium level was the same in the two groups, the hypothesis that the pineal controls or acts with the parathyroid glands is refuted. The massive mobilization of calcium during calciphylaxis may well have obscured some weak pineal function, but the rate of epiphyseal closure should have shown this if it were present, especially in the littermate group of rats where the gland was removed on the day of birth. Any early, transient effect should have been noticeable here, but the X-rays at 90 days showed no difference. This work suggests that the pineal does not play a direct role in calcium metabolism but does not rule out the possibility of an indirect involvement. Calcium values in the *Handbook of Biological Data*[27] were 5.4 to 7.2 mEq. per liter, which is considerably higher than the results reported here. However, we feed our animals strictly on Purina Lab Chow and water and have noticed a tendency for the blood calcium to be low. In a group of fifteen animals given supplemental calcium, the average blood calcium rose to 5.6 mEq. per liter, with extremes of 5.1 to 6.5 mEq. per liter, which would bring these animals in line with the data from the handbook. The blood glucose from the same source is reported as 56 to 76 mg. per 100 cc., which is the same range as our results. The FBS does not seem to be controlled in the rat by the pineal gland.

The determinations selected here fairly well bracket the possible forms of copper found in the bloodstream. Whole blood copper covers that in the red cell as well as the serum. The procedure we have used gives comparable results from one group of animals to another and provides a valid comparison. Two values for blood copper are found in the handbook[27]—1.07 μg. per cubic centimeter and 3.20 μg. per cubic centimeter—and our value of 1.5 μg. per cubic centimeter fits comfortably between them. Plasma includes two forms of copper, that bound into ceruloplasmin and that "free" segment which is loosely attached to the albumin. Ceruloplasmin is a protein with a molecular weight of 151,000 and a dark blue color. It is an enzyme which can use several amines as its substrate, but its primary function, while not definitely known, may well be to deliver copper to the nonexcretory organs.[28] The only known study of ceruloplasmin levels in the rat reported it to be one-third that of the human being,[29] which is refuted by the findings reported here. We find a mean of 41.2 ± 14.8 mg. percent for all the animals in this series, compared with a human mean of 31 mg. percent for males and 36 mg. percent for females according to one report[30] and a mean of 42 mg. percent according to the more recent work.[31] To confirm the validity of the method in our hands, since we had no ceruloplasmin standard, we tested plasma from five normal human beings. These varied from 19.6 to 37.7 mg. percent—well in the reported range.

Each ceruloplasmin molecule contains 8 atoms of copper or 3.2 μg. of copper per milligram of ceruloplasmin.[30] There are 132 μg. of copper per hundred milliliters ($41.2 \times 3.2 = 132$) of rat plasma bound into the ceruloplasmin. If this value is subtracted from the mean total plasma copper (vide supra) of 138 μg. per 100 cc., it yields 6 μg. per 100 cc. of free copper. Methods to determine this value directly have met with failure due to the uncertainty of tying up only nonceruloplasmin-bound copper, so it must be computed by this indirect approach. The free copper—6 μg. per 100 cc.—is 4% of the plasma copper or, stated the other way, the ceruloplasmin copper accounts for 96% of that in the plasma. This is higher than the 90% usually stated for human beings.[32] This and the fact that our plasma copper values are higher than other reports[29,33,34] led us to question our values for rat plasma copper, so we checked them on human plasma. Copper was determined on plasma from eight normal human males and four nonpregnant females. The eight males showed a mean of 1.23 μg. per cubic centimeter, with extremes of 0.94 to 1.73 μg. per cubic centimeter, while the females had a mean of 1.39 μg. per cubic centimeter, with extremes of 1.25 to 1.46 μg. per cubic

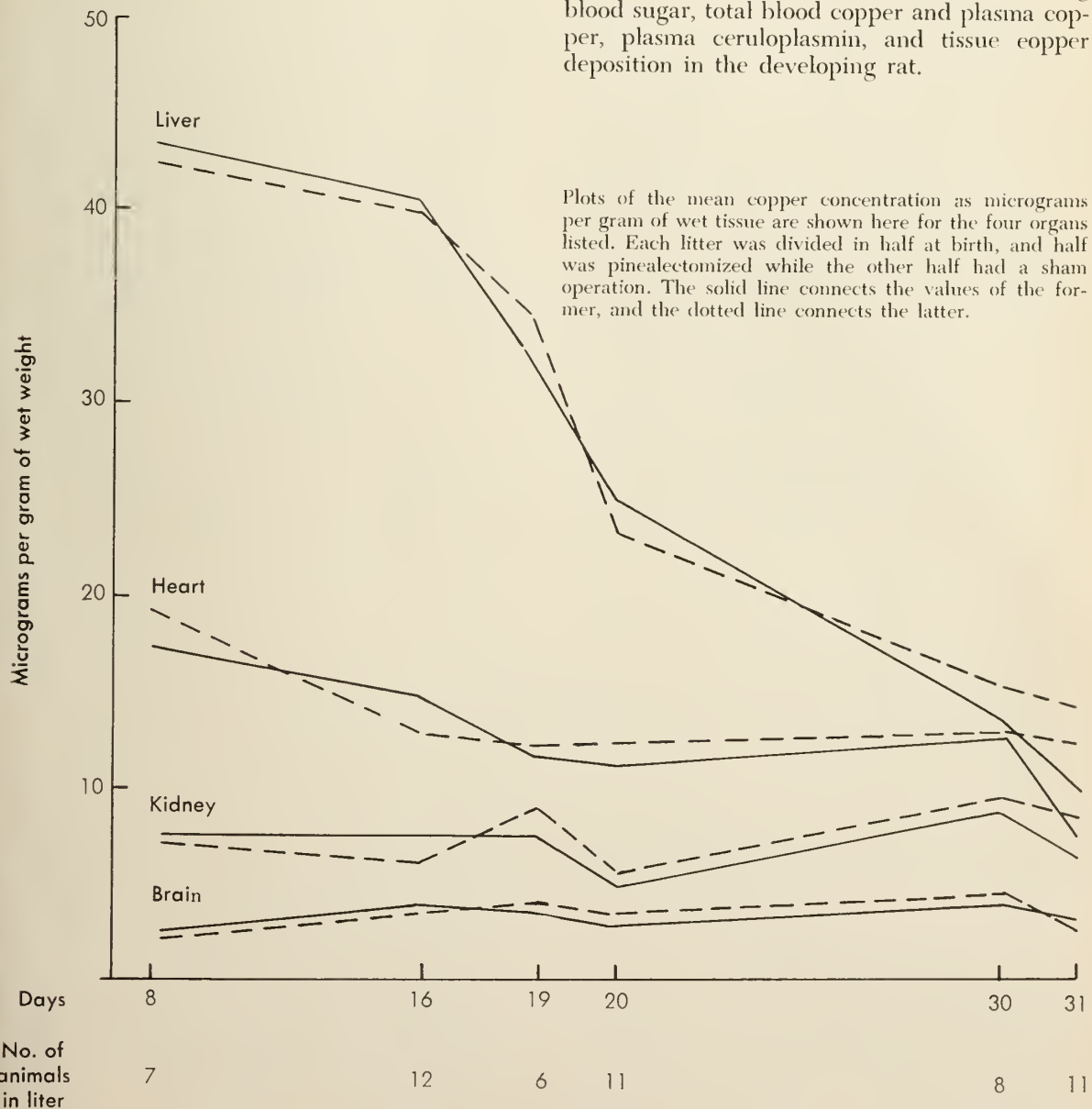
centimeter, which agrees with other reported work[30] and ratifies our results in the rat.

Organ copper deposition was examined in the fetal rat where litter-mate controls were used and the gland excised or the sham operation done on the day of birth. The use of litter mates accounts for the close means (see graph), for organ copper, like most biological variables, varies strongly between litters. Liver copper concentration in the neonatal rat, when the mother receives an adequate copper intake, is about 20 μg. per gram of wet weight on the day of birth, increasing steadily to a high between the sixth and ninth day and decreasing to adult values by the thirtieth day. This pattern of variance of liver copper shown by the young rat has been noted previously[35,36] and is unique among the animals studied.[37] Apparently the neonatal rat is not born with a great reserve of liver copper but, rather, depends on its mother's milk, which is extremely rich in copper,[37] to supply its growth needs.

Pinealectomy at birth certainly doesn't affect this fluctuation of copper in the liver or the steady concentration in the other three organs.

SUMMARY

Normal and pinealectomized rats have been tested in a number of ways, and no significant difference has been found between the two groups. The tests include: calciphylaxis formation, epiphysial closure rate, plasma calcium, fasting blood sugar, total blood copper and plasma copper, plasma ceruloplasmin, and tissue copper deposition in the developing rat.



Plots of the mean copper concentration as micrograms per gram of wet tissue are shown here for the four organs listed. Each litter was divided in half at birth, and half was pinealectomized while the other half had a sham operation. The solid line connects the values of the former, and the dotted line connects the latter.

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Continued from page 466

ORAL ROENTGENOGRAPHIC CHANGES IN WILSON'S DISEASE

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eralized osteoporosis, and old age, it may also be found in the absence of a demonstrable cause.

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Eosinophilic Peritonitis

Report of Two Cases

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EOSINOPHILIC LEUKOCYTOSIS in association with a myriad of clinical conditions has attracted considerable attention. Eosinophilia has been noted in diverse types of infectious processes, in several different cancers, and in numerous collagen diseases or "toxic" conditions, as well as in the more classical parasitic and "allergic" conditions.

Eosinophilic peritonitis, however, has been observed infrequently, despite this massive literature. Two children with eosinophilic peritonitis are reported here because of the variety and the unusual nature of the syndrome. In addition, a short review of experimental knowledge of eosinophilia will be presented.

CASE REPORTS

Case 1. E. W., a 15-year-old white female, noted the onset of dull, aching, but poorly localized lower abdominal pain and a feeling of some abdominal bloating on Nov. 23, 1962. Nausea, vomiting, and diarrhea began shortly after the onset of the abdominal pain, but she remained afebrile throughout her illness. The primary clinical complaint of lower abdominal pain continued and she lost 10 lb. in four weeks.

Past history was entirely unremarkable. She had no allergic history and had never traveled outside the country. Family and social history were entirely unremarkable except for two uncles with asthma.

Physical examination on December 19 revealed an acutely and chronically ill-appearing young female in no severe distress. Vital signs were normal. There was no lymphadenopathy. There was slight distension of the abdomen and a full feeling in the lower abdomen with some mild, scattered tenderness. No abdominal organs or masses were palpable. Pelvic examination showed no tenderness or masses, but there was some fullness in the area of the uterus and adnexa. The remainder of the physical examination was entirely within normal limits.

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Laboratory studies included the following (see table): negative skin tests for tuberculosis, fungi, and trichenella; negative LE clot tests; multiple negative stools for ova and parasites; negative X-rays of the chest, skeletal system, abdomen, upper and lower gastrointestinal tract, and intravenous pyelogram; and negative proctoscopy. A bone marrow was nondiagnostic, showing only an increased number of normal eosinophils.

The abdomen became progressively larger and evidence of ascites became apparent over the next few weeks. The leukocyte count was only slightly elevated this time, with an eosinophilia of 34%. The sedimentation rate was 7 mm. per hour. Laboratory studies included normal electrolytes, BUN, and transaminase, serum proteins were 5.2 gm. percent, and the A/G ratio was 3.1:2.1. Bleeding and clotting times were normal and multiple LE clot tests were negative.

An exploratory laparotomy was performed on Jan. 16, 1963. Muscle and skin biopsies and 700 cc. of almost clear ascitic fluid were obtained. Analysis of the ascitic fluid revealed a protein of 4.6 gm. percent, white count of 11,300 per cubic millimeter with 90% eosinophils, 8% lymphocytes, 2% monocytes, and some reticuloendothelial cells. All cultures were negative. The entire small bowel and colon were completely unremarkable. Lymph node sections showed only chronic lymphadenitis; microscopic sections of the ovary, skin, and muscle were entirely unremarkable. Microscopic examination of the appendix revealed a light infiltrate of cosinophils in the connective tissue, which obliterated the lumen, and in the muscle coats of the appendix. There was a somewhat heavier infiltrate of eosinophils beneath the serosa of the appendix, but there was no evidence of recent inflammation.

This patient did well following surgery and was discharged with a diagnosis of eosinophilic peritonitis. The ascites and eosinophilia remitted without treatment, all abdominal complaints resolved, and the patient has enjoyed good health in the four years since her illness. As in the second case, no specific etiology of the eosinophilia was ever found.

Case 2. E. S., a 6-year-old white male, was admitted to the University of Minnesota Hospitals on Feb. 17, 1966, because of diffuse abdominal pain and significant eosinophilia. Abdominal pain, headache, and lethargy were first noted three days prior to admission. No elevated temperature was ever noted.

Past history was unremarkable. Because of a mild allergic rhinitis he had been skin tested, and only the

BLOOD DATA IN TWO CASES OF EOSINOPHILIC PERITONITIS

Date	Hemoglobin in gm. %	Leukocyte count per cu. mm.	Eosinophils %	Neutrophils %	Sedimentation rate mm. in 1 hr.
12/ 6/62	16	16,800	N	—	—
12/11/62	Unchanged	12,700	43	32	—
12/19/62	15.7	20,250	68	20	—
1/15/63	N	10,250	34	27	7
Peritoneal fluid:		11,300	90	0	—
1966	N	N	4	N	N

(N) = normal

Date	Hemoglobin in gm. %	Leukocyte count per cu. mm.	Eosinophils %	Neutrophils %	Sedimentation rate mm. in 1 hr.
4/65	N	N	8	42	N
11/65	N	N	19	62	—
2/16/66	N	60,000	70	17	—
2/17/66	11.2	48,200	82	9	76
2/24/66	N	10,300	23	38	86
2/26/66	N	10,000	17	45	—
3/ 9/66	N	6,200	9	50	53
4/ 6/66	N	8,600	16	39	37
5/25/66	N	6,400	10	58	25
11/15/66	12.4	11,900	23	36	23

(N) = normal

patch test to house dust was positive. He had had no recent clinical symptoms of allergic rhinitis. He had had a right orchiopexy and herniorrhaphy ten months prior to admission; a complete blood count at this time showed a normal leukocyte count with 8% eosinophils. Family and social history were unremarkable. On physical examination he was a pale, slender boy in no distress and with normal vital signs. The tonsils were markedly enlarged, without inflammation. Diffuse anterior and posterior cervical adenopathy was noted; the nodes ranged in size from 0.5 to 1.5 cm. in diameter and were firm and tender. Moderate epigastric tenderness to palpation was noted. The remainder of the physical examination was entirely negative.

Hemoglobin was 11.2 gm. percent. White cells numbered 48,000 per cubic millimeter with 10% neutrophils, 7% lymphocytes, 1% monocytes, and 82% eosinophils (see table). The eosinophils were noted to be larger in size than usual. The platelet count was normal. The sedimentation rate was 76 mm. per hour. The total serum proteins were 7.9 gm. percent, A/G ratio was 3.6:4.3, and serum protein electrophoresis was unremarkable except for an elevated alpha-2 globulin. Serum immunoelectrophoresis and tests for delayed hypersensitivity were normal. Routine laboratory data included the following normal determinations: urinalysis, BUN, fasting blood sugar, serum electrolytes, calcium, and phosphorus. LE clot tests, VDRL, and fluorescent antinuclear antibody tests were all negative. Direct Coombs, partial thromboplastin time, and liver function tests were also within normal limits. Chest roentgenogram and skeletal survey were entirely normal.

Fresh stool specimens were examined for ova and parasites and were negative on four occasions. Tuberculin, fungal, and Trichinella skin tests were negative. An upper gastrointestinal series and intravenous pyelogram were entirely negative.

A cervical lymph node biopsy revealed benign lymphoid hyperplasia. A closed biopsy of the liver was entirely normal. Bone marrow examination revealed a marked eosinophilia of 57%, but the majority of these cells were mature forms and no striking abnormalities were seen; the marrow otherwise was entirely unremarkable.

He remained well except for the mild abdominal pain which persisted for several months. As shown in the table, his peripheral blood had returned to normal in one month except for the elevated sedimentation rate, which decreased three months after discharge. Nine months after discharge, the patient continued well except for occasional episodes of tonsillitis. A mild leukocytosis and eosinophilia of 23% were still present and the sedimentation rate remained at 20 mm. per hour. He had no allergic difficulties whatsoever, and no cause for his eosinophilic leukemoid reaction has been found as of this date.

DISCUSSION

Patients in whom blood eosinophilia is associated with any of a variety of gastrointestinal symptoms, such as abdominal pain, distension, or ascites, may well have eosinophilic peritonitis. This often occurs as a subtle lesion associated with infiltration of the serosa of the hollow viscera in the absence of mechanical obstruction and can be recognized only by microscopic examination of the appropriate tissues. The leukocyte count may range from normal up to 26,000, and the blood eosinophilia is only rarely less than 1,500 per cubic millimeter.[1] Pain may be the only abdominal complaint. Although ascites is very common, it does not occur in every patient.

One of the cases reported by Harley had abdominal pain, vomiting, diarrhea, and an episode of ascites with 90% eosinophils. Two of his reported cases had an eosinophilic peritonitis of the small intestine, and the other two demonstrated an eosinophilic peritonitis of the appendix. Only two of the cases had liver biopsies; one of these showed conspicuous eosinophils in the sinusoids. The circumstantial evidence provided by these and other cases suggests that the eosinophilic infiltration of the gastrointestinal tract was responsible for the symptoms. No surgical procedure or therapeutic agent was uniformly successful and steroids were of only short-term benefit when used. Surgical intervention was of no value and the authors concluded that surgery should be avoided. The microscopic sections of the involved peritoneum in all the cases demonstrated a necrotizing angiitis of the hypersensitivity type involving the affected serosal vessels, thus suggesting an allergic etiology.

However, they were unable to demonstrate an allergic basis for eosinophilic peritonitis and concluded that it was as yet an entity of unknown etiology, usually self-limited and of an episodic nature. The clinical picture of our first case clearly resembles the cases described by Harley. Our second case with blood eosinophilia and persistent abdominal pain could also well represent eosinophilic peritonitis, for cases without ascites do occur. The percutaneous needle biopsy of the liver was normal and was the only abdominal tissue examined.

Another abdominal condition associated with blood eosinophilia is eosinophilic granuloma of the gastrointestinal tract.[2] Many of the patients demonstrate a significant blood eosinophilia sometime during their illness and the abdominal symptoms can mimic peptic ulcer, neoplasm, biliary tract disease, or pyloric obstruction. The histologic picture consists of eosinophilic infiltrations which, unlike eosinophilic peritonitis, are primarily in the submucosa. However, the muscular layers and serosa may also be involved, and there is a tendency to perivascular infiltration of the eosinophils. Unlike the eosinophilic granulomas of bone and soft tissue, which contain histiocytes and reticulum cells, these eosinophilic granulomatous infiltrates of the gastrointestinal tract contain predominantly eosinophils. Further, while the eosinophils are densely accumulated in small groups in the former, they are more evenly distributed in the latter. Although malignancy has not been demonstrated in these lesions, surgery is usually required to make the distinction even if the location of the lesion itself does not require resection.

Archer presents a wealth of information to support his belief that released histamine is the common denominator in most, if not all, clinical conditions associated wtih blood eosinophilia.[3,4] Since the eosinophils are attracted to released histamine, there is a tendency for these cells to congregate in areas which have higher and higher histamine concentrations. When the histamine concentration of an organ exceeds that of the blood passing through it, therefore, one would expect retention of some eosinophils in that organ. A similar argument applies to release of eosinophils from the bone marrow into the blood when the blood histamine level is greater than that of the marrow. This concept, of course, refers only to concentrations of released histamine. Histamine contained within mast cells, for example, has no effect unless released. At the present time, we know very little about the actual mode of action of eosinophils, except that when histamine is exposed to eosinophils, the histamine is changed. This effect has no bearing on the therapeutic use of the synthetic antihistamines, for the effect of eosinophils is not competition for receptor sites in tissue. The effect of eosinophils, therefore, differs from both the corticosteroids and the antihistamines; in a sense, it is midway between them but is clearly separate from both because it is the only one of the three in which histamine itself is changed.

Most of the conditions associated with blood eosinophilia can be explained by the above hypothesis. Patients with a significant helminth infestation, for example, have an eosinophilia of both blood and bone marrow which is approximately equivalent. Migration of larvae produces a tissue reaction with significant numbers of eosinophils present, and one can demonstrate cellular infiltrations along the tracks of migrating larvae. The most important mechanism of this eosinophilia, according to Archer, is local histamine release in tissue secondary to damage caused by the parasites. This damage can be prolonged or chronic and can therefore provide an eosinotactic effect on the blood for long periods of time. If the blood histamine is elevated, development of eosinophils in the bone marrow is also stimulated. If the parasites are antigenic to the host, then histamine will also be released as a result of the antigen-antibody reaction.

The pathogenesis of eosinophilia in allergic states, as in parasitic diseases, can be related to histamine release, in this case in the region affected by the allergen. This histamine release, secondary to the antigen-antibody reaction, attracts eosinophils, often in large enough numbers to produce a local cosinophilia. There is no blood eosinophilia, but there indeed may be blood eosinopenia if the allergic reaction leads to shock, for the increased levels of circulating corticoids will suppress eosinophil release from the marrow. In patients with asthma, or allergic rhinitis, the sputum, or nasal smear, may contain many eosinophils without a simultaneous blood eosinophilia—it is the eosinophilia of the respective tissue, lung or nasal mucosa, which is the essential feature of the allergic condition.

If the hypothesis presented by Archer is correct, eosinophilic leukemoid reactions such as occurred in our two patients should be associated with increased blood levels of histamine. Unfortunately, no appropriate studies on our two patients were performed. In patients in whom the eosinophilic leukemoid reaction represents a disorder of cellular metabolism, i. e., leukemia, however, one would not necessarily expect that a disorder in histamine metabolism was operative.

Despite the wealth of information available to support the hypothesis that histamine will account for most eosinophilic phenomena, this is not universally accepted. For example, if it is assumed that serum from patients with a high systemic eosinophilia contains a factor like histamine which has attracted them into the blood stream, then passive transfer of such sera should induce an eosinophil response in the recipient. Attempts at inducing such a response have uniformly failed.

Litt has performed a number of studies on the induction of eosinophilia and the function of eosinophils.[5–7] He concurs with the findings of Archer and others that eosinophils contain histamine and may transport it to the site of an allergic reaction and that eosinophils may also contain an antihistamine which inactivates histamine at the site of an allergic reaction. He has also shown that there is a direct correlation between the number of eosinophils and the amount of histamine in an exudate. Litt believes, however, that histamine does not play an important role in mediating tissue eosinophilia because it cannot produce the degree of eosinophilia commonly seen in clinical and experimental situations.[8] His studies have shown that the eosinophil is a very early participant in the response of lymph nodes to encounter with an antigen. A substance appears to be generated in the lymph nodes which causes the eosinophils to emigrate from the blood stream and temporarily accumulate perifollicularly. This localization resembles that of antibody-containing plasma cells but occurs a few days earlier. He reasons that since this type of response to primary antigenic stimulation is unique to lymphoid tissue and since it occurs in other tissue only when antibody is present, the lymph node may well be contributing the required antibody. Eosinophils would, therefore, be cells which are exquisitely sensitive detectors of antibody complex.

But yet another paradox that needs explanation is the frequency of eosinophilia in diverse immune deficiency states. In Bruton type agammaglobulinemia, in Swiss type agammaglobulinemia, and in anergic conditions as Hodgkin's disease or Aldrich syndrome, a marked eosinophilic leukocytosis is part of the clinical picture. If antigen-antibody complex were significant in the clinical sense, then the eosinophilia in the above diseases requires further thought.

The eosinophil's capability of phagocytosis has been well demonstrated. This cannot be a very important function, however, since the eosinophil is conspicuously absent from acute microbial infections, presumably secondary to steroid mechanisms. Studies by Litt have demonstrated that eosinophils can injest soluble antigen-antibody complexes. There is thus circumstantial evidence that a similar event can occur in a variety of pathological states, including allergy. Since eosinophilia can be induced with common antigens and ordinary precipitating antibody, it would seem that eosinophilia reflects a general class of events, of which allergy is a subclass. In short, eosinophilia would not be a specific indicator of allergy alone but rather a reflection of the presence of antigen-antibody complexes of various types.

CONCLUSIONS

Eosinophilic peritonitis in two children has been presented. Abdominal pain and marked eosinophilic leukocytosis were the common characteristics. The 15-year-old girl developed transitory ascites, and exploratory laparotomy revealed the characteristic eosinophilic infiltrates in the appendix. The 6-year-old boy had symptoms and physical signs of peritoneal irritation. Despite intensive investigation of both patients no specific etiology could be noted.

The theories of eosinophilia and their relationship to abdominal pain are discussed. The histamine (Archer) theory and the antigen-antibody complex concept (Litt) are outlined.

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Carcinoma of the Prostate: A Review

Part I. Incidence, Etiology, Pathology, and Diagnosis

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ADENOCARCINOMA OF THE PROSTATE comprises between 10 and 20% of all cancers in the adult male.[1] According to the United States Public Health Monograph No. 56, 1959, the average age for all stages of prostatic cancer is 70 years.[2] Carcinoma of the prostate is uncommon in men less than 50 years of age. At the State University of Iowa Hospitals, only 1% of all cases of prostatic cancer occurred in patients less than 50 years of age.[3] The annual incidence of adenocarcinoma of the prostate ranges between 35 and 40 cases per 100,000 total population, whereas the annual mortality rate from prostatic cancer is approximately 8 per 100,000 total population or 17 per 100,000 male population. This makes carcinoma of the prostate the third highest ranking cause of death from cancer in adult males, exceeded only by carcinoma of the stomach and bronchogenic carcinoma.[2] It is an interesting observation that the incidence of prostatic carcinoma is slightly higher in Negroes than in Caucasians but is much lower in Orientals.

In various autopsy series the incidence of adenocarcinoma of the prostate ranges between 14 and 46%, depending upon the age group studied, the number of serial sections taken from the prostate, and the histological criteria used in diagnosing early carcinoma of the prostate.[4–6] When the step-section rather than the random-section technique of examining the prostate is used, the average incidence of prostatic cancer in men over 50 years of age as determined at necropsy is 24%. When the random-section technique is used in studying tissue removed by transurethral resection from clinically benign prostate glands at the Mayo Clinic, the incidence of unsuspected carcinoma of the prostate is approximately 13%.[7] The incidence of prostatic cancer should be higher and approach the 24% autopsy incidence if more pieces are examined and if the step-section technique is employed.[8]

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ETIOLOGY

Like benign nodular hyperplasia, adenocarcinoma of the prostate is probably caused by a hormonal imbalance incident to the process of aging. Although excess androgen stimulation has been incriminated, there have been no verified case reports of prostatic carcinoma caused by the prolonged administration of androgens either to man or to the experimental animal. Huggins and Hodges,[9] nevertheless, have demonstrated that approximately 70% of patients with prostatic cancer have tumors which initially respond to changes in their hormonal environment.

PATHOLOGY

Mostofi[10] has stated that there are approximately thirty histological features characteristic of adenocarcinoma of the prostate. Paradoxically, any single characteristic may be, but is not always, diagnostic of prostatic cancer. The microscopic appearance is variable, depending upon the histological grade. In general, carcinoma of the prostate is characterized by a disorderly pattern of the prostatic acini which tend to be quite small and closely packed together, the so-called back-to-back arrangement (Fig. 1). Cellular and nuclear pleomorphism, hyperchromatism, and increased mitotic activity may be lacking. Perineural invasion, which is found in 80 to 90% of cases, confirms the diagnosis of carcinoma of the prostate (Fig. 2). Recent investigations have

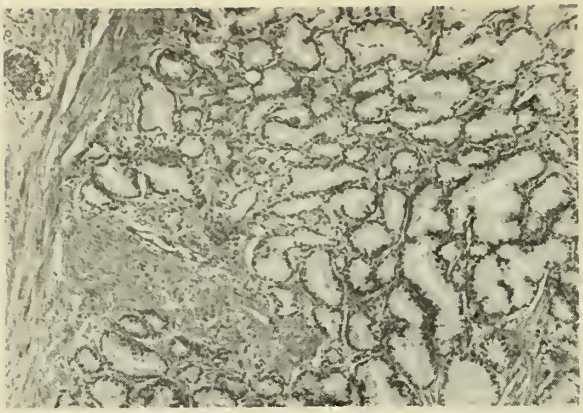


Fig. 1. Photomicrograph demonstrating well-differentiated adenocarcinoma of prostate

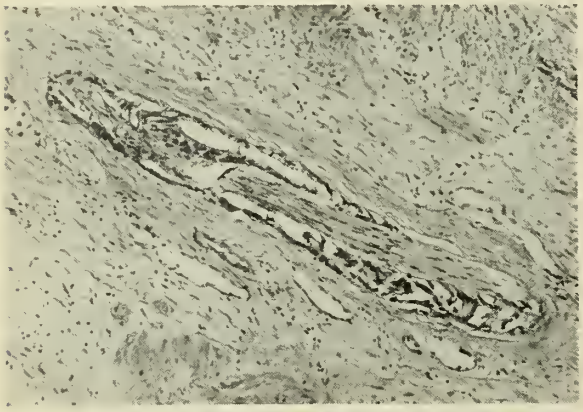


Fig. 2. Photomicrograph demonstrating perineural invasion by prostatic carcinoma

shown that perineural lymphatics do not actually exist but are in reality only perineural spaces.[11] Invasion of these spaces occurs early and is of diagnostic rather than of prostognostic significance.[12] The more advanced to anaplastic carcinomas of the prostate show more cellular dedifferentiation and less of a tendency to form acini (Fig. 3). A cribriform pattern and signet-ring cell formation are variations in the histological picture of prostatic cancer. Mellinger and associates[13] have recently described a new system of determining a patient's prognosis by combining the histological grade with the clinical stage of his prostatic carcinoma.

It was originally thought that adenocarcinoma of the prostate began as a single, small focus in the posterior lobe or lamella of the prostate. By means of step-section examination of the entire prostate, it was recently discovered that prostatic cancer is usually multicentric in origin and may begin in any one or several of the prostatic lobes.[1] As previously mentioned, carcinoma of the prostate involves the perineural spaces early in the course of the disease. Since these perineural spaces are not true lymphatic channels, the finding of perineural invasion does not necessarily denote the presence of lymphogenous spread. The microscopic focus of prostatic carcinoma eventually grows to a size where it can be palpated on rectal examination. Exactly how long this takes can only be surmised, but it is thought to take many years and undoubtedly varies from one patient to the next. The small cancerous nodule is usually located in the periphery of the prostate far removed from the prostatic urethra. Early nodules, therefore, seldom cause obstructive symptoms and usually remain clinically silent. The carcinoma continues to infiltrate, most likely through the perineural spaces, until the anatomical capsule of the prostate is invaded. Once the capsule is penetrated, metastases to the regional lymph nodes may promptly occur. The cancer may continue to spread until the fascial coverings of the seminal vesicles, the bladder neck, and the prostatic and membranous portions of the urethra are involved. The two layers of Denonvilliers' fascia which separate the prostate from the rectum are seldom penetrated by carcinoma of the prostate. Invasion of the prostate by rectal cancer, on the other hand, is not uncommon.

When microscopic invasion of the anatomical capsule of the prostate is found alone, the prognosis is not necessarily bad and the regional pelvic lymph nodes are seldom involved by metastases. Arduino and Glucksman[14] found that when the seminal vesicles are free of cancer and no other local extension of the tumor is found, the pelvic lymph nodes are involved in only 7% of the cases. When the seminal vesicles are microscopically invaded by carcinoma, however, there is an 82% or higher incidence of pelvic lymph node metastases. Arduino is of the opin-

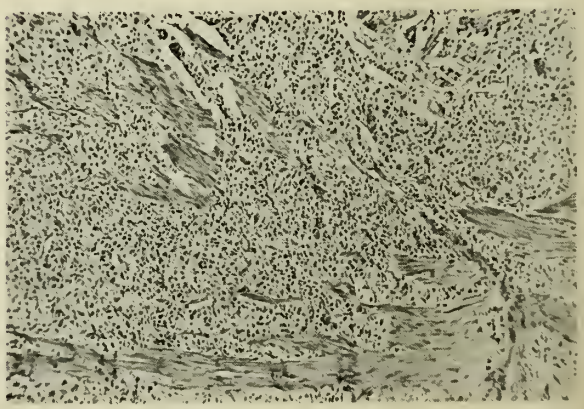


Fig. 3. Photomicrograph demonstrating poorly differentiated adenocarcinoma of prostate

ion that when local extension of the prostatic cancer is clinically evident, the incidence of lymph node metastases approaches 100%. From the regional pelvic lymph nodes—primarily the internal iliac nodes—prostatic carcinoma can metastasize to the periaortic nodes and, occasionally, to the cervical and supraclavicular nodes. Rarely, the inguinal lymph nodes are involved.

Dissemination of adenocarcinoma of the prostate to the skeletal system is fairly common. The venous drainage of the prostate as described by Beneventi and Noback[15] is primarily into the internal iliac veins. Beneventi demonstrated not only free communication between the pelvic veins and the vertebral plexus of veins as described by Batson[16] but also to many other parts of the body. The predilection of metastases for the lumbosacral spine and pelvis is most likely explained by this communication between the pelvic and vertebral venous plexuses. The exact role of the lymphatics in disseminating prostatic carcinoma to bone is equivocal. The spread of cancer cells through perineural lymphatics to the lumbosacral spine and pelvis has been postulated by Warren and associates[17] as another pathway of tumor dissemination. More recently, lymphangiography in patients with metastatic carcinoma of the prostate has demonstrated a distribution of lymphatic vessels in the lumbosacral and pelvic regions which pursues a course similar to that of the vertebral plexus of veins. Should the regional pelvic lymph nodes become blocked by tumor, it is possible that cancer cells are diverted from their usual lymphatic channels and are carried through these alternate lymphatics which invest the bony pelvis and vertebrae together with the vertebral plexus of veins.[18] Furthermore, lymphangiography has demonstrated direct communications between lymphatics and veins in the pelvis. Other commonly affected parts of the skeleton are the ribs, the femurs, and the dorsal and cervical vertebrae. Skeletal metastases from prostatic carcinoma are usually osteoblastic and produce a characteristic picture which will be described later. Osteolytic lesions are occasionally seen and may occur along with osteoblastic metastases.

When carcinoma of the prostate metastasizes to other organs, the lungs and liver are most often involved.

SYMPTOMS AND PHYSICAL FINDINGS

Early carcinoma of the prostate seldom produces symptoms. A clinically unsuspected, microscopic focus or foci of prostatic carcinoma may be associated with benign nodular hyperplasia and discovered incidentally by the pathologist in an enucleated adenoma or in one or two fragments of prostate removed by transurethral resection. Often the first sign of prostatic cancer is a small, hard nodule confined to the prostate and discovered on routine digital-rectal examination. Because of its usual peripheral location in the prostate, the small nodule seldom causes obstructive symptoms. More advanced carcinoma of the prostate may cause prostatism and urinary retention. Creevy[19] estimates that prostatic cancer accounts for 15 to 20% of prostatic obstructions. By this time the induration as felt on rectal examination has usually extended beyond the confines of the prostatic capsule and has involved the region of the seminal vesicles. Hematuria occurs in patients with carcinoma of the prostate but is more common in men with benign nodular hyperplasia. Upward extension of the prostatic carcinoma from the region of the bladder neck and seminal vesicles occasionally causes hydronephrosis if one or both distal ureters are compressed by neoplasm.

The patient generally feels well until the cancer metastasizes. He may then complain of weakness, a poor appetite, and weight loss and is often found to be anemic. The latter results from involvement of the bone marrow by the cancer. The patient may first seek medical advice because of back or hip pain, a pathological fracture, or neurological symptoms due to skeletal metastases or nerve root compression. The bone pain experienced by the patient with metastatic carcinoma of the prostate is usually deep, continuous, and severe. Metastases to the inguinal, cervical, and supraclavicular lymph nodes are occasionally uncovered on physical examination. Edema of one or both lower extremities is usually due to obstruction of the pelvic lymphatics by metastatic carcinoma. Carcinoma of the prostate will occasionally invade the rectum and cause rectal pain, obstruction, and bleeding.

DIAGNOSIS

A proper approach to the diagnosis of prostatic carcinoma should include a biopsy of any suspicious lesion in the prostate for histological confirmation and clinical staging of the cancer once a positive tissue diagnosis has been made.

Prostatic biopsy. Other prostatic lesions which may simulate carcinoma include prostatitis with subsequent fibrosis of the prostate, benign nodular hyperplasia, nonspecific granulomatous prostatitis, tuberculous prostatitis, infarct of the prostate, and prostatic calculi. The latter may coexist with prostatic cancer. When a suspicious area is palpated on rectal examination of the

prostate, the only certain method of making a diagnosis is by biopsy. Digital-rectal examination is only 50% accurate when small nodules or localized areas of induration are encountered.[20] For reasons to be mentioned later, treatment should be withheld until a positive tissue diagnosis has been made. Methods of prostatic biopsy include transurethral biopsy, transrectal biopsy, retropubic biopsy, and closed and open perineal biopsy.

Transurethral biopsy is usually not adequate for diagnosis of early carcinoma of the prostate. As mentioned earlier, the small nodule or localized area of induration representing early carcinoma is usually located in the peripheral portion of the prostate far removed from the prostatic urethra. If only one or two pieces of prostate removed by transurethral resection for benign nodular hyperplasia show carcinoma, it is still possible that the cancer is confined to the prostate. If the majority of resected pieces demonstrate carcinoma, the cancer is most likely far advanced locally and not suitable for extirpative surgery.

Grabstald and Elliott[21] popularized the transrectal prostatic biopsy in this country. The reason a few urologists prefer the transrectal approach is that the prostatic nodule usually lies just beneath the rectal mucosa and is therefore more accessible, and the biopsy needle can be inserted perpendicular to the nodule rather than tangentially through the perineum. A prostatic nodule can also be exposed through a small incision in the rectal wall and a specimen removed with biopsy forceps. The bowel is always prepared before biopsy with enemas and neomycin or a minimally absorbed sulfonamide. While proponents of the procedure claim an accuracy equal to or higher than that of closed perineal biopsy, opponents of the method object to passing a biopsy needle from a contaminated area such as the rectal lumen into a presumably sterile area such as the prostate. In addition, transrectal biopsy could make subsequent extirpative surgery more difficult.

The accuracy of the retropubic biopsy should approach that of the open perineal biopsy. Since most cancers of the prostate are palpable on the posterior surface of the gland, the prostate in the retropubic approach must be elevated from the rectum and rotated in order to expose the lesion for biopsy. The main advantage of this approach is that it allows inspection of the iliac lymph nodes for metastases.

Closed perineal biopsy of the prostate using the Vim-Silverman or one of the other biopsy needles is quite popular but does have a few limitations. In cases where there is no palpable nodule or induration, only a very small portion of the total volume of the prostate can be sampled. In their famous Bowery series, Hudson and associates[22] performed both transperineal needle biopsy and open perineal biopsy on 200 consecutive men with palpably normal prostate glands. In 179 cases both the needle biopsy and the open biopsy were benign. The open perineal biopsy demonstrated prostatic carcinoma in 21 patients, whereas the needle biopsy disclosed cancer in only 2 of these cases. The transperineal needle biopsy is more accurate when a small nodule or area of induration can be palpated, but considerable skill is required in directing the point of the needle into the lesion. Also, the core of tissue removed is relatively small. When the efficacy of needle biopsy is compared to that of open perineal biopsy of the prostate containing a small palpable lesion, the needle biopsy was found to be approximately 70% accurate.[23] Needle biopsy of prostatic lesions is of value only if the diagnosis is positive for cancer. A negative tissue diagnosis does not rule out carcinoma. If the index of suspicion is still high after two separate, negative needle biopsies, some urologists will proceed with an open perineal biopsy of the prostate. The highest degree of accuracy is obtained by needle biopsy of locally far-advanced prostatic cancers.

Needle biopsy of the prostate may be an office procedure but is generally performed in a hospital using either a local or a general anesthesia. One type of biopsy needle, which is quite popular, is the Franklin modification of the Vim-Silverman needle (Fig. 4). The technique of transperineal needle biopsy of the prostate will be briefly described (Fig. 5). The patient is placed on the operating table in the lithotomy position and the perineum is prepared with a suitable antiseptic solution. If the patient has not voided completely prior to the biopsy, he is catheterized and the bladder is emptied. The biopsy needle is advanced through the perineal skin anterior to the anus and is guided by a finger in the rectum to the suspicious prostatic lesion. The tip of the needle remains anterior

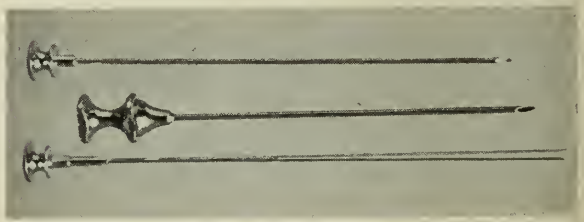


Fig. 4. Franklin modification of Vim-Silverman biopsy needle

to the rectal wall where it can easily be palpated and directed. After the tip of the biopsy needle has been guided to the lesion, the obturator is removed and the cutting blades are inserted into the sheath. The cutting blades are then introduced into the suspicious area and the sheath is advanced over the blades, thereby trapping a cylinder of tissue within the latter. When the Franklin modification of the Vim-Silverman needle is used, the needle does not have to be rotated in order to free the core of tissue. The cutting blades along with the encased biopsy specimen are removed, and the sheath is left in place in the event additional specimens are desired. Mellinger[1] prefers to obtain at least five specimens at the time of needle biopsy. The most common complication of needle biopsy of the prostate is mild, transient hematuria due to penetration of the bladder or prostatic urethra. A rare complication of needle biopsy is the implantation of cancer cells along the needle tract.[24–26]

In the hope of finding more men with early, surgically curable carcinoma of the prostate, Hudson[27] performed an open perineal biopsy on 686 Bowery men who had no palpable prostatic lesions suggestive of cancer. Some of these men had minimal prostatic hypertrophy, whereas the remainder had no complaints referable to the prostate. A tissue diagnosis of adenocarcinoma of the prostate was made in 76 of the 686 men (11%) in whom an open perineal biopsy was performed. The tumor was found to be inoperable in 9 of the 76 cases (12%). In 23 cases in which the first biopsy was reported as benign, a subsequent biopsy uncovered carcinoma in 3 (13%). Open perineal biopsy seems to be an impractical method of diagnosing early prostatic cancer when it is in the microscopic or latent stage. When the prostate feels normal, the urologist has no idea of what part of the gland to biopsy. If the 24% autopsy incidence of latent carcinoma in aging males is applicable to the Bowery series, then the open perineal biopsy is less than 50% accurate in diagnosing microscopic or latent carcinoma of the prostate. In addition, the clinical importance of microscopic carcinoma of the prostate must be considered. Opinion is divided between those who consider it as having a high and those who consider it as having a low lethal potentiality. Open perineal biopsy is most valuable in those cases where a small prostatic nodule or area of induration is palpable; under these circumstances, it should approach 100% accuracy.[28]

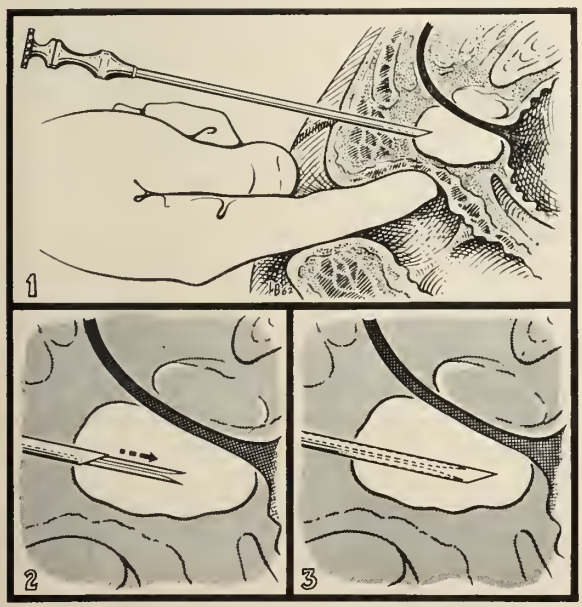


Fig. 5. Technique of transperineal needle biopsy of prostate: [1] Tip of biopsy needle has been guided to suspicious area in prostate, [2] obturator has been removed and cutting blades have been introduced into lesion, and [3] sheath has been advanced over cutting blades, thereby trapping a cylinder of tissue.

Cytological studies. Cytological studies of the urine and prostatic secretions have not proved to be as accurate in detecting early carcinoma of the prostate as was originally desired. Cytological study of the prostatic smear appears to be more useful as an adjunct to the diagnosis of more advanced neoplasms of the prostate than as a screening procedure for early prostatic cancer.[29] There has been some reluctance among urologists to massage a possibly cancerous prostate in the fear of disseminating malignant cells.

Before the prostatic cancer can be properly staged, the presence or absence of metastases must be determined. Laboratory and X-ray studies utilized for this purpose include the determination of the serum acid and alkaline phosphatase, the skeletal survey, the intravenous urogram, and the bone marrow aspiration. Cystourethroscopic examination may be helpful in the clinical staging of prostatic carcinoma. Newer tests designed to determine the presence of metastatic disease will be mentioned.

Acid phosphatase. The serum acid phosphatase is a very important determination in establishing the presence of metastatic carcinoma of the prostate. Acid phosphatase is an enzyme that hydrolyzes organic phosphoric esters liberating inorganic phosphate in a pH range of 4.0 to 6.0. A wide variety of normal tissues contain acid phosphatase in high concentrations. These include the prostate, erythrocytes, liver, kidneys, and spleen. In the prostate the enzyme is elabor-

ated by the glandular epithelial cells.[30] Kutscher and Wolbergs[31] in 1935 were the first to discover acid phosphatase in the prostate. In 1937 Bodansky[32] published a method for determining acid phosphatase by using beta-glycerophosphate as a substrate buffered to about pH 5.0. In 1938 Gutman and Gutman[33] observed an increase in the serum acid phosphatase in some patients with disseminated prostatic carcinoma. These investigators had previously noted that cancerous prostatic tissue contained large amounts of the enzyme.[34] Gutman and Gutman[35] subsequently adapted the King-Armstrong method for measuring serum alkaline phosphatase to the determination of the serum acid phosphatase utilizing phenylphosphate as a substrate also buffered to around pH 5.0. In most laboratories the upper limits of normal for the two tests are 1 Bodansky unit and 4 King-Armstrong units.

Manipulation of the normal or hyperplastic prostate may transiently elevate the serum acid phosphatase, with the values usually returning to normal within twenty-four hours.[36] Hemolysis of a clotted blood sample may also falsely elevate the total serum acid phosphatase by releasing the enzyme from erythrocytes. Certain diseases other than disseminated prostatic carcinoma have been reported in rare instances to elevate the serum acid phosphatase. These conditions include various primary and metastatic diseases of the bone; certain metabolic, renal, and liver disorders; and diseases involving the reticuloendothelial and hematopoietic systems.[37] Excluding these exceptions, an elevated serum acid phosphatase signifies that the prostatic cancer has disseminated and can no longer be cured by radical prostatectomy—total prostatoseminal vesiculectomy. Normally, the acid phosphatase secreted by the prostatic epithelial cells is transported through the prostatic ducts to the prostatic urethra. Very little of the enzyme reaches the bloodstream, but it is found in the prostatic secretion in high concentrations. When the cancer cells elaborating the enzyme metastasize to the lymph nodes, bone, or other organs, the acid phosphatase can no longer be carried to the urethra through the prostatic ducts, but instead passes into the circulation causing an elevation of the serum acid phosphatase. While the serum acid phosphatase is rarely elevated in the absence of disseminated prostatic cancer, it may be normal in the presence of metastatic disease. It is estimated that the serum acid phosphatase is normal in 20 to 30% of patients with osseous metastases and in 65 to 75% of cases with soft tissue metastases.[38]

In an effort to devise a biochemical test diagnostic of early prostatic carcinoma, refinements in the acid phosphatase determination were made beginning with the discovery by Abul-Fadl and King[39] in 1949 that there were differences in the acid phosphatase derived from different tissues. Fishman and Lerner[40] subsequently developed a method of determining the prostatic fraction of the serum acid phosphatase utilizing the discovery that L-tartrate inhibits the prostatic acid phosphatase but does not affect the enzyme in the erythrocytes. In most laboratories the upper limit of normal for the prostatic serum acid phosphatase is 1 King-Armstrong unit. Investigators hoped that prostatic fraction determinations would be of value as a screening test in detecting cancer of the prostate before it broke through the confines of the prostatic capsule. According to Clarke,[41] the prostatic fraction is never elevated in patients with carcinoma localized to the prostate, and according to Mellinger,[1] it is never elevated with strictly local extension beyond the prostatic capsule. Opinion is divided as to the sensitivity of the prostatic serum acid phosphatase in detecting early metastatic carcinoma of the prostate and in the follow-up evaluation of these patients.

A simple and reliable method of shipping a serum for acid phosphatase determination has been devised by Dr. Richard Doe and associates[42] at the Veterans Administration Hospital in Minneapolis, Minn. It was discovered that the prostatic serum acid phosphatase values could be preserved at room temperature up to one week by mixing the patient's serum with measured numbers of disodium-citrate buffer tablets. This makes it possible for the general practitioner living in a rural area to ship a serum to a central laboratory and obtain a reliable serum acid phosphatase determination.

Alkaline phosphatase. Whereas the serum acid phosphatase is rarely elevated in diseases other than metastatic cancer of the prostate, the serum alkaline phosphatase is frequently elevated in such disorders as intrahepatic and extrahepatic biliary obstruction, Paget's disease, primary bone tumors, metastatic cancer to bone, and metabolic diseases affecting bone.[43] Even though the serum alkaline phosphatase has been reported to be elevated in as high as 86% of patients with metastatic carcinoma of the prostate, the test is still not specific for metastatic disease.[44]

Skeletal survey. Cancer of the prostate commonly metastasizes to the lumbosacral spine and pelvis, followed in order of frequency by metastases to the ribs, the femurs, and the other vertebrae. A kidney, ureter, and bladder X-ray along with a separate X-ray of the pelvis which in-



Fig. 6. X-ray of pelvis demonstrating osteoblastic metastases from adenocarcinoma of prostate

cludes the proximal femurs will usually be sufficient to detect the majority of demonstrable osseous metastases. Characteristically, the lesions are osteoblastic, appearing as single or multiple, fairly well-circumscribed areas of increased density, usually with loss of the normal trabecular pattern (Fig. 6). In 10% of cases with osseous metastases, osteolytic lesions are noted (Fig. 7). Osteolytic lesions are occasionally mixed with osteoblastic areas (Fig. 8). Paget's disease may produce radiological changes quite similar to those of metastatic carcinoma of the prostate. In Paget's disease there is increased density of bone, but the normal trabecular pattern is retained and there may be subperiosteal deposition of bone (Fig. 9). The bones most frequently involved in osteitis deformans are the pelvis, femurs, and skull.

Intravenous urogram. The intravenous urogram may be helpful in the clinical staging of the prostatic cancer patient only if ureteral obstruction can de demonstrated. If the cancer has extended from the region of the bladder neck and seminal vesicles upward to involve one or both distal ureters, the carcinoma can no longer be completely removed by radical prostatectomy.

Cystourethroscopic examination. If radical prostatectomy is contemplated, cystourethroscopic examination should be performed. If the carcinoma has invaded the membranous urethra or trigone of the bladder, it is unlikely that total prostatoseminal vesiculectomy will completely eradicate the cancer. In more advanced cases of prostatic carcinoma, cystourethroscopy may be indicated if the patient is having symptoms of prostatism.

Bone marrow aspiration. Bone marrow aspiration is a useful adjunct in the diagnosis of disseminated prostatic cancer. On a few occasions metastatic carcinoma of the prostate will be suspected even though the total and prostatic serum acid phosphatase determinations are normal and



Fig. 7. X-ray of femur demonstrating osteolytic metastases from adenocarcinoma of prostate



Fig. 8. X-ray of pelvis demonstrating mixed osteoblastic and osteolytic metastases from adenocarcinoma of prostate

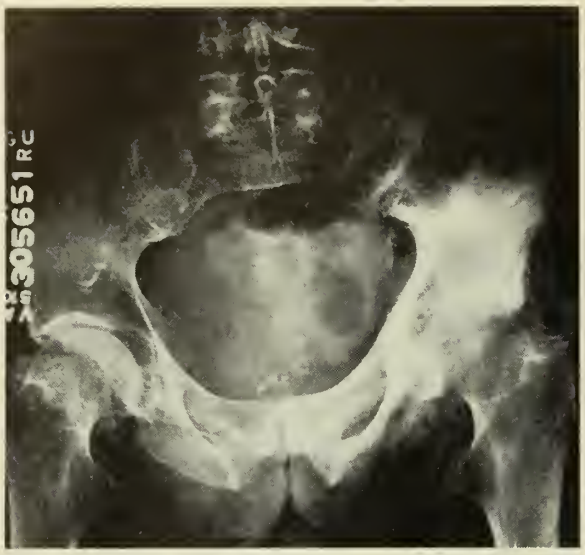


Fig. 9. X-ray of pelvis demonstrating Paget's disease

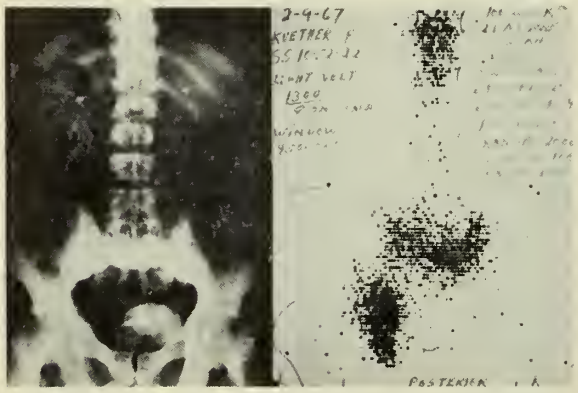


Fig. 10. Intravenous urogram (left) demonstrating osteoblastic metastases in spine and pelvis. Strontium 85 photoscan (right) showing increased radioactivity in areas corresponding to osteoblastic lesions

neither osteoblastic nor osteolytic lesions are demonstrable on X-ray. Bone marrow obtained from the sternum or iliac crests may demonstrate tumor cells in patients on whom the biochemical and roentgenographic studies are negative.[45] Approximately one-half of patients with clinically detectable prostatic cancer have positive bone marrow biopsies.[45,46] Mehan and associates[47] noted a 22% incidence while Flocks[48] noted only a 10% incidence of malignant marrow cells in patients with prostatic cancer. All of these authors agree that bone marrow aspiration should be performed as part of the routine evaluation of patients being considered for radical prostatectomy.

Lactic dehydrogenase. Denis and Prout[49] discovered that the total serum lactic dehydrogenase (LDH) is at least borderline, if not actually elevated, in most treated or untreated patients with progressively active prostatic carcinoma. The total serum LDH, however, is not specific for carcinoma of the prostate. These investigators had previously demonstrated a marked increase in one of the isozymes of LDH (LDH V) in prostatic cancerous tissue. In patients with progressively active carcinoma, elevations in the serum of the isozymes LDH IV and LDH V were demonstrated. Patients with prostatic cancer in remission had only traces of these isozymes. No quantitative relationship between isozymes LDH IV and LDH V and the serum acid phosphatase could be demonstrated. In the future this test may be of some value in detecting impending relapse in treated patients.

It is the consensus of most investigators that the urinary LDH determination is of little value in diagnosis of prostatic carcinoma.[50]

Lactate:citrate ratio in prostatic tissue. Cooper and Farid[51] have reported that biochemical changes may take place in the prostate for a considerable period of time before the diagnosis of cancer can be made histologically. They found high concentrations of citrate in benign nodular hyperplasia, whereas in cancer of the prostate the citrate concentration was markedly reduced or absent. Lactate, however, was fairly uniform in concentration in both benign and malignant prostatic tissue. In benign nodular hyperplasia the average lactate:citrate ratio was 0:6, whereas in carcinoma the average ratio was approximately 1:0. In advanced prostatic carcinoma the values for the lactate:citrate ratio were considerably higher. It is possible that in the near future biochemical assay of prostatic biopsy tissue will detect an increasing number of very early cases of prostatic cancer.

Strontium 85 photoscan. Strontium, like calcium, is incorporated into areas of increased osteoblastic activity. Simpson and Orange[52] and, more recently, Williams and Blahd[53] have reported on the diagnostic value of total body photoscanning seventy-two hours after the administration of strontium 85 to patients with clinically proven adenocarcinoma of the prostate (Fig. 10). These investigators feel that the strontium 85 scan is superior to the serum acid phosphatase determination, the skeletal survey, and the bone marrow aspiration in ruling out metastases. Like the latter examination, the strontium 85 photoscan may be a valuable adjunct in the evaluation of patients being considerd for total prostatoseminal vesiculectomy.

Part II on treatment will appear in a subsequent issue.

Henry L. Williams, Jr., M.D.

JEROME A. HILGER, M.D.
St. Paul



BECAUSE OF HIS SCHOLARLY ATTRIBUTES, Dr. Henry L. Williams, Jr., has been a thoroughly good influence on younger men in medicine, particularly those in otolaryngology. His outstanding position in American otolaryngology has given his views and attitudes a wide hearing. He has combined a thorough historical knowledge with a searching and often caustic common sense. His interests and fund of information are as wide as medicine itself. He has been the great "leavener" in the world of otolaryngology—bringing to a broad specialty the yeast of general medical fundamentals without which specialty medicine deteriorates into devitalizing isolated compartmentation.

The combination of sound basic medical knowledge with a frank, and often impersonally brutal, willingness to "discuss" has made for an awesome reputation for this man of medicine. Essayists beware when he seems to nap. Any levity in the viewers disappears at discussion time. From a seeming narcoleptic state he has been known to rise in midtheater, shuffle to the podium, and distress a shallow presentation with basic truths of significance greater than that of the essay itself. He customarily leaves the program enriched beyond the primary offering. Certainly no others in the field of otolaryngology, and few in the whole spectrum of medicine, have done more to maintain the vigorous, impersonal, and learned tone of medical discussion.

Dr. Henry L. Williams, Jr., was born in Philadelphia in 1898. His physician father had been a distinguished Yale athlete who subsequently shaped an unusual double career in athletics and medicine. Dr. Williams, Sr., was a specialist in obstetrics and gynecology. In the world of athletics he was a football mentor of major prowess. From West Point, where he first coached, he moved to Philadelphia to coach at the University of Pennsylvania. In 1900 he accepted the post of football coach at the University of Minnesota. Here he continued his dual role until he retired from athletics in 1921. His original thought and fundamental contribution earned him a permanent place in the football "Hall of Fame" and established Minnesota as a force in modern football. The Williams Arena at the University of Minnesota was named to honor him as the originator of modern football in the North Central United States.

A tale is told of Dr. Williams, Sr., that may shed some light on the characteristic of individualism this Englishman passed on to his son. When it was officially decided (over the obstetrician coach's vehement objections) that intercollegiate football participants would be identified by jersey numbers, his compliance was modified by an original never-say-die contribution. His Minnesota players were fielded with jerseys bearing a long string of numerals much like a railroad freight car.

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This is an introduction to a group of papers on otolaryngology to be published as the Henry L. Williams, Jr., Series.

Henry L. Williams, Jr., after his boyhood years in Minneapolis, returned to Philadelphia for his secondary education at Penn Charter School. He returned thence for a year at the University of Minnesota, where he earned a letter in football in 1917 and, undoubtedly, bettered himself to some extent academically. Thereafter, he attended Yale University for three years to qualify for his Bachelor of Arts degree. His rangy physique and athletic capability resulted in stellar performances on the Yale track team as a hurdler.

In medical school at the University of Pennsylvania there was less opportunity for athletics. By the time of his graduate training in otolaryngology at the Mayo Clinic in Rochester subsequent to internship at St. Agnes Hospital in Philadelphia, his athletic pursuits had veered toward a knowledgeable spectator interest, but his active participation in outdoor activities has never waned. In these, ranging from golf to hunting, he has had the double pleasure reserved to those who undertake each pursuit in scholarly fashion, applying some principles and ignoring others in the manner of one who participates primarily to enjoy. It has been said that his duck call produces more banter than birds. The long sweep of his trout fly is typically that of one with long-frame leverage abetted by an extra long rod. The cast is for distance—a joy with or without the strike. As a matter of fact, for him, the tail gate of the station wagon covered with gear can provide the complete expedition without approach to the water.

Dr. Williams completed his formal training program in otolaryngology at the Mayo Clinic in 1928. He attained board certification in otolaryngology in 1930. The American Board of Otolaryngology was then in its sixth year of service to medicine—a time when board certification did not yet exist in such major medical fields as general surgery and internal medicine.

Dr. Williams assumed a staff position in the Section of Otology and Rhinology at the Mayo Clinic in 1928. His year of greatest good fortune was 1930 when he married Helen Craddock, a native of Pine Island, Minn., then working in Rochester. They subsequently enlarged their family to five with the addition of three children.

From 1932 to 1934 "Bill" practiced otolaryngology in Minneapolis. These depression years were lean in material goods but rich in friendship values, which have continued to the present.

Dr. Williams returned to the Mayo staff in 1934. His experiences and studies over the next ten years backgrounded thirty-six important papers on tympanomastoid disease and nasal sinus disease with particular reference to the recognition and management of their dreadful complications. During these years, he established a solid reputation for diagnostic acumen and sound surgery and his fearsome image in medical discussion was born.

World War II occasioned another interruption in his service at the Mayo Clinic. At an age when many physicians felt no compulsion to participate, Dr. Williams served in the Navy Medical Corps as lieutenant commander until 1943.

In the postwar years, Dr. Williams became one of the best-known teachers of otolaryngology in the United States. He participated in most sections of the country in continuation educational and postgraduate programs. He published the most comprehensive monograph on Meniere's disease, fundamental papers on the intrinsic allergic state, and dissertations on head pain, chemotherapy, otosclerosis, sinal and tympanomastoid surgical techniques, endolymphatic hydrops, autonomic dysfunction, lethal granuloma, facial paralysis, auditory testing, collagen disease, neoplasia, vertigo, nasal pyramid surgery, and neuro-otologic diagnosis. His cosmopolitan interests were exceeded only by the depths of his analysis. His contributions number more than one hundred.

In 1949, Dr. Williams succeeded his longtime associate, Dr. Harold Lillie, as head of the Section of Otology and Rhinology at the Mayo Clinic. He continued in this authority until 1962, when he became senior consultant to the section. In true character this change occasioned him to several ribald story allusions to the essence of "senior consultant" status.

Retirement from the Mayo Clinic at the established age to a life of inactivity was an impossible prospect for this active scholar. It was the good fortune of the Department of Otolaryngology at the University of Minnesota that Dr. Williams joined the department as chief of the affiliate service at the Veterans Administration Hospital at Fort Snelling. His reservoir of knowledge, experience, and technique has been at the service of the graduate training program and the patients to the present time. The graduate men have, in addition, had the rich experience of association with a truly inspiring scholar, vigorous discusser, and well-humored friend.

It is safe to say that the next retirement of Dr. Henry Williams, Jr., will lead to yet a third career. His reservoir of fact and hypothesis has never been more full, and his willingness to share it is as generous as ever. One hopes the North Central States conferences will continue to benefit from his participation.

INDEX TO VOLUME 87

January 1967 through December 1967

SUBJECT INDEX

Aging and aged, lung cancer, diagnosis (Levine & Geller) 97 (Mar.)

Allergy, to oxacillin without known sensitivity to penicillin (case report, Peters et al.) 10 (Jan.)

Allopurinol for gout (Seebach) 411 (Oct.)

Amputation
- leg, psychological aspects (Simpson & Albronda) 429 (Nov.)
- phantom limb phenomenon, effect of LSD on mechanism (Kuromaru et al.) 22 (Jan.)

Aorta, abdominal, ruptured aneurysm, roentgenographic features (Nevin) 259 (July)

Apparatus
- artificial heart-lung machine, present status (Rossi) 89 (Mar.)
- internal pacemaker for complete heart block associated with pregnancy (case report, Berestka & Spellacy) 461 (Dec.)

Artery, pulmonary, atresia in nondisabled young adult (case report, Al-Attar) 102 (Mar.)

Arthritis
- rheumatoid, physical rehabilitation (symposium, Martin) 112 (Apr.)
- rheumatoid, role of physical therapy (symposium, Stillwell) 133 (Apr.)

B

Bacteria, bacteremia due to gram-negative bacilli, management (Martin) 439 (Nov.)

Balance, problems in restoration after central nervous system damage (symposium, Kottke) 197 (June)

Biographies
- Anderson, Gaylord W., M.D. (Thomson) 30 (Jan.)
- Anderson, John A., M.D. (Wannamaker) 270 (July)
- Anderson, Karl W., M.D. (Opstad) 421 (Oct.)
- Baker, A. B., M.D. (Shapiro) 105 (Mar.)
- Krusen, Frank H., M.D. (Eckman) 140 (Apr.)
- Tovell, Ralph Moore, M.D., (Lundy) 291 (Aug.)
- Wangensteen, Owen H., M.D. (Myers) 216 (June)
- Williams, Henry L., Jr., M.D. (Hilger) 485 (Dec.)

Biopsy, prostate, disposable perineal needle for (Mellinger) 103 (Mar.)

Bladder
- incontinence in men, correction with Berry prosthesis and silastic balloon (Hoskins & Mellinger) 268 (July)
- problems with myelopathies (symposium, Schutt et al.) 187 (June)
- vesicoureteral reflux, ureteral reimplantation for, results (Creevy) 238 (July)

Blood
- eosinophilia, tropical pulmonary, clinical, hematologic, and radiologic features (Narang) 457 (Nov.)
- eosinophilic peritonitis (case reports, Hunt et al.) 473 (Dec.)
- transfusions in factor VIII deficiency (Dana) 143 (May)

Blood vessels, cerebral vasospasm in migraine (Dalessio) 283 (Aug.)

Bones
- osteogenesis imperfecta, pregnancy with (case report, Dunham & Spellacy) 293 (Aug.)
- osteoporosis, diagnosis and treatment (symposium, Cole) 118 (Apr.)

BOOK REVIEWS
- Abramson (ed.): Resuscitation of the Newborn Infant and Related Emergency Procedures: Principles and Practice 29A (Jan.)
- Adams (ed.): Current Practice of Orthopaedic Surgery, 1966 25A (Mar.)
- Adriani (ed.): Appraisal of Current Concepts in Anesthesiology 14A (July)
- Allen (ed.): Radiology in World War II 14A (Aug.)
- Andrew: One World of Science: Personal Visits to Men of Research in Many Lands 31A (Apr.)
- Ansfield: Chemotherapy of Disseminated Solid Tumors 11A (Oct.)
- Bargeon: Reticuloendothelioses in Childhood: A Clinical Survey 14A (Aug.)
- Blades (ed.): Surgical Diseases of the Chest 14A (June)
- Boba: Death in the Operating Room 29 (Jan.)
- Boyd: Spontaneous Regression of Cancer 32A (Jan.)
- Burstein: Fundamental Considerations in Anesthesia 29 (Jan.)
- Bykov & Kurtsin: International Series of Monographs in Cerebrovisceral and Behavioral Physiology and Conditioned Reflexes, Vol. 2: The Corticovisceral Theory of the Pathogenesis of Peptic Ulcer 31A (Feb.)

Calman: Atlas of Hernia Repair 24A (Apr.)

Cameron: Hyaluronidase and Cancer 31A (Jan.)

Cassels & Ziegler (eds): Electrocardiography in Infants and Children 41A (Jan.)

Croll & Brady (eds.): Recent Advances in Nuclear Medicine: Based on a Symposium sponsored by the Department of Radiology of the Hahnemann Medical College 33A (Feb.)

Dahlin: Bone Tumors 12A (Sept.)

Davis & Landau: Clinical Capillary Microscopy 18A (Mar.)

DeReuck & Knight (eds.): Ciba Foundation Symposium: Touch, Heat and Pain 292 (Aug.)

Eastman & Hellman: Williams Obstetrics 35A (Jan.)

Eckenhoff (ed.): Science and Practice in Anesthesia 28 (Jan.)

Fahrni: Backache Relieved Through New Concepts of Posture 31A (Jan.)

Field: Soviet Socialized Medicine: An Introduction 13A (Aug.)

Fredericks & Morse (eds.): Anesthesia for Open Heart Surgery 29 (Jan.)

Gabe: International Series of Monographs in Pure and Applied Biology/Zoology Division: Neurosecretion 29A (Jan.)

Gallagher: Medical Care of the Adolescent 31A (Apr.)

Giannestras: Foot Disorders: Medical and Surgical Management 12A (Oct.)

Goss (ed.): Gray's Anatomy: Anatomy of the Human Body 22A (Mar.)

Gray: Pulmonary Embolism 17A (May)

Hamby: Carotid-Cavernous Fistula 27A (Apr.)

Hollander: Arthritis and Allied Conditions 42A (Jan.)

Jackson: The Cervical Syndrome 17A (Aug.)

Joint Group of International Federation of Gynaecology and Obstetrics and International Confederation of Midwives: Maternity Care in the World: International Survey of Midwifery Practice and Training 32A (Feb.)

Juret: Endocrine Surgery in Human Cancers 29A (Jan.)

Knock: Anticancer Agents 13A (Aug.)

Kolodny & McLoughlin: Comprehensive Approach to Therapy of Pain 292 (Aug.)

Lasagna (ed.): International Encyclopedia of Pharmacology and Therapeutics, Section 6, Volumes I and II: Clinical Pharmacology 12A (Oct.)

Leigh & Marley: Bronchial Asthma: A Genetic, Population and Psychiatric Study 12A (Sept.)

LeNoir: Congenital Idiopathic Talipes 14A (July)

Leopold (ed.): Ocular Therapy: Complications and Management 17A (May)

Lim: Cardiopulmonary Function Tests in Clinical Medicine 11A (July)

Lorian: Antibiotics and Chemotherapeutic Agents in Clinical and Laboratory Practice 24A (Apr.)

Masserman (ed.): Science and Psychoanalysis, Vol. X: Sexuality of Women 11A (July)

Mark & Papper (eds.): Advances in Anesthesiology: Muscle Relaxants 11A (Oct.)

Nagel: The Mayo Legacy 29 (Jan.)

Nahum (ed.): Early Management of Acute Trauma 29A (Feb.)

Neil & Warren: Vertebrate Biochemistry 40A (Jan.)

Ney & Friedenberg: Radiographic Atlas of the Genitourinary System 25A (Apr.)

Norman (ed.): Cardiac Surgery 12A (Oct.)

Pearson: Historical and Experimental Approaches to Modern Resuscitation 28 (Jan.)

Pergamon Press: Radiation Protection: Recommendations of the International Commission on Radiological Protection (Adopted September 17, 1965) 11A (June)

Pfeutze & Radner (eds): Clinical Tuberculosis: Essentials of Diagnosis and Treatment 30A (Feb.)

Pittinger: Hyperbaric Oxygenation 28 (Jan.)

Powell: Comprehensive Background for Anesthesiology 29 (Jan.)

Prasad (ed.): Zinc Metabolism 18A (Mar.)

Prevot: Monograph of the Pasteur Institute: Manual for the Classification and Determination of the Anaerobic Bacteria 26A (Apr.)

Quick: Hemorrhagic Diseases and Thrombosis 32A (Jan.)

Raab (ed.): Prevention of Ischemic Heart Disease: Principles and Practice 11A (July)

Rosenbloom: The Practice of Cystoscopy 30A (Feb.)

Schade: The Peripheral Nervous System 11A (June)

Scott & Dodd: Neurological and Neurosurgical Nursing: An Introduction 14A (Aug.)

Segre (ed.): Androgens, Virilization and the Hirsute Female 12A (Sept.)

Smith: Blood Diseases in Infancy and Childhood 35A (Jan.)

Steiner: Physician-Generals in the Civil War: A Study in Nineteenth Mid-Century American Medicine 39A (Jan.)

Stephens & Stanfield (eds.): Cellular Concepts of Rheumatoid Arthritis: Holbrook Memorial Symposium 17A (May)

Thompson et al. (eds.): The Stomach (Including Related Areas in the Esophagus and Duodenum): The Thirteenth Hahnemann Symposium 11A (June)

Todd: The Commonwealth and International Library of Science Technology Engineering and Liberal Studies: Health and Humanity: An Introduction to Medicine 32A (Feb.)

Van Auken (ed.): The Artful Practice of Medicine 11A (June)

Volle: International Encyclopedia of Pharmacology and Therapeutics, Section 12: Ganglionic Blocking and Stimulating Agents: Vol. 1: Muscarinic and Nicotinic Stimulant Actions at Autonomic Ganglia 16A (Aug.)

Wershub: One Hundred Years of Medical Progress: A History of the New York Medical College Flower and Fifth Avenue Hospitals 11A (Oct.)

Wiltse: United States Army in World War II: The Medical Department: Medical Service in the Mediterranean and Minor Theaters 42A (Jan.)

Winqvist (ed.): Comparative Leukaemia Research: Proceedings of an International Wenner-Gren Symposium held in Stockholm, September 1965 27A (Apr.)

Ziegler: Cardiac Evaluation in Normal Infants 31A (Feb.)

Zimmerman (ed.): Intravascular Catheterization 32A (Jan.)

Bowel problems with myelopathies (symposium, Schutt et al.) 187 (June)

Boynton series see Ruth E. Boynton Series

Brain

cerebral abscess and cyanotic congenital heart disease (case reports, Calkins & Bell) 403 (Oct.)

internal hydrocephalus, results from shunting procedures (Christoferson) 321 (Sept.)

Bronchi, acute bronchiolitis as cause of chronic obstructive lung disease in adults (Dines) 281 (Aug.)

C

Calcium metabolism in rat, relation to pineal gland (Wiederanders & Evans) 467 (Dec.)

Cancer

hemangiopericytoma with pulmonary metastasis (case report, Bogedain & Carpathios) 213 (June)

lung, primary, diagnosis in elderly (Levine & Geller) 97 (Mar.)

metastatic, role of physical therapy (symposium, Martin) 112 (Apr.)

pain control (Owens) 287 (Aug.)

prostatic, review of incidence, etiology, pathology, and diagnosis (Blackard) 477 (Dec.)

Carbohydrate metabolism, effect of alteration on vitamin C state (Setyaadmadja et al.) 18 (Jan.)

Cerebrovascular disorders, rehabilitation, practical aspects (symposium, Martin) 112 (Apr.)

Chondritis, development after chest or abdominal surgery (Lewis) 247 (July)

Circulation

pulmonary artery atresia in nondisabled young adult (case report, Al-Attar) 102 (Mar.)

pump oxygenator, present status (Rossi) 89 (Mar.)

Coma, hyperglycemic nonketotic, induced by peritoneal dialysis (Whang) 453 (Nov.)

Copper metabolism in rat, relation to pineal gland (Wiederanders & Evans) 467 (Dec.)

Cyclophosphamide for chronic, steroid-resistant glomerular disease in children (Campbell & Jacinto) 149 (May)

Cysts, renal, differentiation from tumors (Mellinger) 432 (Nov.)

Cytoxan® (cyclophosphamide) for chronic, steroid-resistant glomerular disease in children (Campbell & Jacinto) 149 (May)

D

Demineralization, oral, in Wilson's disease (Ogle et al.) 464 (Dec.)

Demyelinizing diseases, physical rehabilitation, benefits (symposium, Martin) 112 (Apr.)

Dialysis, peritoneal, hyperglycemic nonketotic coma due to (Whang) 453 (Nov.)

Diet, role in obesity (Wilson et al.) 13 (Jan.)

Drugs

causing iatrogenic disorders (Olin) 390 (Oct.)

combined immunosuppressive, for chronic, steroid resistant glomerular diseases in children (Campbell & Jacinto) 149 (May)

penicillins, characteristics and uses (Martin) 79 (Mar.)

E

Ecthyma, contagious, in man (case reports, Lambie & Gustafson) 400 (Oct.)

Electroencephalograms, responses to sound and light in infants (Engel) 181 (May)

Emphysema, physical therapy (symposium, Martin) 112 (Apr.)

Epilepsy, classification and treatment in children (Copple & Isom) 167 (May)

Eskimos, health, role of Department of National Health and Welfare (Coodin) 51 (Feb.)

Esophagus, esophagopleural fistula complicating lobectomy (case report, Konsuwan & Chatikavanich) 450 (Nov.)

Exercise, role in obesity (Wilson et al.) 13 (Jan.)

Eye, strabismus, surgical management (Reed & Bigourdan) 49 (Feb.)

F

Forensic medicine, legal definition of incompetency (O'Toole) 273 (Aug.)

G

Gout, allopurinol treatment, indications and results (Seebach) 411 (Oct.)

H

H. D. LEES Series

Institutional environmental health and safety: the Minnesota story (Michaelsen) 1 (Jan.)

Hearing, objective evaluation by microphonic techniques (Long et al.) 251 (July)

Heart

arrest, resuscitation instruction in hospital by anesthesia department (McCaughey) 46 (Feb.)

atrial septal defects, diagnostic features (Hamilton et al.) 385 (Oct.)

complete block, as complication of acute myocardial infarction, transvenous pacemaker treatment (Johanson & Guenter) 393 (Oct.)

complete block, associated with pregnancy and treated with internal pacemaker (case report, Berestka & Spellacy) 461 (Dec.)

cyanotic congenital disease and cerebral abscess (case reports, Calkins & Bell) 403 (Oct.)

failure in infancy, treatment (Lees & Way) 178 (May)
patent ductus arteriosus, experience with surgical corrections (Campbell et al.) 415 (Oct.)
rheumatic disease, diagnosis and treatment (Lucas) 230 (July)
two-chambered right ventricle with primary pulmonary hypertension (case report, Golwalla et al.) 210 (June)

Heart surgery, hemodynamics during heart-lung bypass (Rossi) 89 (Mar.)

Hemiplegia
perceptual disorders in (symposium, Sawtell & Martin) 193 (June)
physical treatment (symposium, Bjorklund) 123 (Apr.)

Hemophilia, transfusion therapy (Dana) 143 (May)

Hypertension, pulmonary, two-chambered right ventricle with (case report, Golwalla et al.) 210 (June)

Hypoglycemia, neonatal, symptoms and treatment (Haworth & McRae) 41 (Feb.)

I

Iatrogenic disorders, relation to medical progress (Olin) 390 (Oct.)

Infarction, myocardial, acute, complicated by complete heart block, transvenous pacemaker treatment (Johanson & Guenter) 393 (Oct.)

Infections, contagious ecthyma in man (case reports, Lambie & Gustafson) 400 (Oct.)

Influenza, type B, 1967 incidence in USSR (Zhdanov) 419 (Oct.)

Institutions
Child Development Clinic of Children's Hospital of Winnipeg, collaborative treatment (Bell et al.) 68 (Feb.)
Children's Hospital, Winnipeg, program (Medovy) 34 (Feb.)
St. Paul–Ramsey County Hospital, dedication address (Wangensteen) 73 (Mar.)
University of Manitoba, pediatric program (Medovy) 34 (Feb.)
University of Minnesota Health Service, environmental health and safety program (Michaelsen) 1 (Jan.)
Winnipeg Rh laboratory, history (Chown) 38 (Feb.)

Instruments, disposable needle for perineal biopsy of prostate (Mellinger) 103 (Mar.)

Intestines
problems with myelopathies (symposium, Schutt et al.) 187 (June)
small bowel examination with routine stomach studies, techniques (Gilchrist) 236 (July)

J

Joint disease, degenerative, practical rehabilitation (symposium, Martin) 112 (Apr.)

K

Kidney, chronic steroid resistant glomerular disease in children, combined immunosuppressive therapy (Campbell & Jacinto) 149 (May)

L

Lees, H. D., see H. D. Lees Series

Leg, amputation, psychologic aspects (Simpson & Albronda) 429 (Nov.)

LSD, effect on phantom limb phenomenon, mechanism (Kuromaru et al.) 22 (Jan.)

Lung
cancer see Cancer, lung
chronic obstructive disease in adults, acute bronchiolitis as cause (Dines) 281 (Aug.)
esophagopleural fistula complicating lobectomy (case report, Konsuwan & Chatikavanich) 450 (Nov.)
mediastinal abscess of subdiaphragmatic origin (Nickerson et al.) 265 (July)
obstructive disorders, physical therapy for (symposium, Halpern) 125 (Apr.)
pulmonary artery atresia in nondisabled young adult (case report, Al-Attar) 102 (Mar.)
pump oxygenator, present status (Rossi) 89 (Mar.)
tropical pulmonary eosinophilia, clinical, hematologic, and radiologic features (Narang) 457 (Nov.)

M

Medical profession, physical medicine and rehabilitation as a specialty (symposium, Kottke) 109 (Apr.)

Metabolism
copper and calcium in rat, relation to pineal gland (Wiederanders & Evans) 467 (Dec.)
during heart-lung bypass (Rossi) 89 (Mar.)

Migraine, cerebral vasospasm in (Dalessio) 283 (Aug.)

Mouth
abnormalities in Wilson's disease (Ogle et al.) 464 (Dec.)
cleft lip, palate, dental aspects (Cleall) 64 (Feb.)
cleft lip, surgical correction (Baird) 57 (Feb.)
cleft lip, team approach in treatment (Kernahan et al.) 55 (Feb.)
cleft palate, role of speech therapist in treatment team (Vines) 62 (Feb.)
cleft palate, speech after operation, secondary procedures (Waters) 60 (Feb.)
cleft palate, team approach in treatment (Kernahan et al.) 55 (Feb.)

Myxedema after removal of ectopic thyroid resembling thyroglossal duct cyst (case report, Saiki) 7 (Jan.)

N

Nerve section for relief of cancer pain (Owens) 287 (Aug.)

Nervous system, central
restoration of balance after injury (symposium, Kottke) 197 (June)
Wilson's disease, oral roentgenographic changes 464 (Dec.)

Newborn infants
electroencephalographic responses to sound and light (Engel) 181 (May)
illness in, physiologic basis for supportive management (Behrman) 157 (May)
imminent death, due to enlarged thymus gland (case report, Shannon) 78 (Mar.)
reflex responses for determining gestational age (Babson & McKinnon) 174 (May)

North Dakota State Medical Association
transactions 296 (Aug.); 333 (Sept.)
Woman's Auxiliary membership roster 425 (Oct.)

O

Obesity, causes and perpetuation, preventive measures (Wilson et al.) 13 (Jan.)

Osteoporosis, diagnosis and treatment (symposium, Cole) 118 (Apr.)

OWEN H. WANGENSTEEN Series
Owen H. Wangensteen (Myers) 216 (June)

Oxacillin, allergic reaction without known sensitivity to penicillin (case report, Peters et al.) 10 (Jan.)

P

Pacemakers
internal, for complete heart block associated with pregnancy (case report, Berestka & Spellacy) 461 (Dec.)
transvenous, for acute myocardial infarction complicated by complete heart block (Johanson & Guenter) 393 (Oct.)

PAIN AND DISTRESS Section
Cerebral vasospasm in migraine (Dalessio) 283 (Aug.)
Control of pain in patients with cancer (Owens) 287 (Aug.)
Dr. Ralph Moore Tovell (Lundy) 291 (Aug.)
Effect of LSD on the phantom limb phenomenon (Kuromaru et al.) 22 (Jan.)
Mystery of atypical pain (Lundy) 283 (Aug.)

Paraplegia, mobility with, techniques and devices to aid patients (symposium, Hallin) 130 (Apr.)

Parkinson's syndrome, treatment and rehabilitation (symposium, Martin) 112 (Apr.)

PEDIATRICS Special Issues
Children's Hospital of Winnipeg 33-72 (Feb.)
An integrated approach to cleft lip and palate (Kernahan et al.) 55
Child Development Clinic of The Children's Hospital and its method of collaborative treatment (Bell et al.) 68
Cleft lip (Baird) 57
Dental aspects of the cleft lip and palate (Cleall) 64
Introduction (Grant) 33
Neonatal hypoglycemia: a six-year experience (Haworth & McRae) 41
Pediatrics in Manitoba: goals and accomplishments (Medovy) 34
Role of the speech therapist on the cleft palate team (Vines) 62
Some observations on Eskimo health (Coodin) 51
Strabismus: review of 100 surgical cases (Reed & Bigourdan) 49
Teaching resuscitation in a children's hospital (McCaughey) 46
The story of the Winnipeg Rh laboratory (Chown) 38
Treatment of cleft palate (Waters) 60

University of Oregon 143-181 (May)
Classification and treatment of seizure disorders in children (Copple & Isom) 167
Combined immunosuppressive therapy in steroid-resistant, chronic glomerular diseases of childhood (Campbell & Jacinto) 149
Determination of gestational age in premature infants (Babson & McKinnon) 174
Electroencephalographic responses to sound and to light in premature and full term neonates (Engel) 181
Heart failure in infancy (Lees & Way) 178
Phenylketonuria: report of the Oregon detection and evaluation program (Stewart & Ashley) 162
Physiologic basis for the supportive management of the sick newborn (Behrman) 157
Prefatory remarks (Olmsted) 143
Transfusion therapy in factor VIII deficiency (Dana) 143

Penicillins, characteristics and uses (Martin) 79 (Mar.)

Peritoneum, eosinophilic peritonitis (case reports, Hunt et al.) 473 (Dec.)

Phenylketonuria, report of Oregon detection and evaluation program (Stewart & Ashley) 162 (May)

PHYSICAL MEDICINE AND REHABILITATION Symposium
Early history of physical medicine and rehabilitation at the University of Minnesota (Knapp) 136 (Apr.)
Frank H. Krusen, M.D.: pathmarker in physical medicine and rehabilitation (Eckman) 140 (Apr.)
History of physical medicine at the University of Minnesota from 1945 to 1965 (Olson) 203 (June)
Osteoporosis (Cole) 118 (Apr.)
Perceptual problems of the hemiplegic patient (Sawtell & Martin) 193 (June)
Physical medicine and rehabilitation as a medical specialty (Kottke) 109 (Apr.)
Physical medicine for patients with rheumatic disease (Stillwell) 133 (Apr.)
Physical therapy in obstructive pulmonary disease (Halpern) 125 (Apr.)
Physical treatment for the hemiplegic patient (Bjorklund) 123 (Apr.)
Physiology of balance (Kottke) 197 (June)
Practical rehabilitation (Martin) 112 (Apr.)

Problems of bladder and bowel in patients with myelopathies (Schutt et al.) 187 (June)
Relationship of the staff physician to the department of physical medicine and rehabilitation of a community hospital (Knapp) 207 (June)
Transfers and mobility for paraplegic and quadriplegic patients (Hallin) 130 (Apr.)

Physicians
relationship of staff physician to department of physical medicine and rehabilitation of community hospital (Knapp) 207 (June)
surgical training, St. Paul–Ramsey County Hospital, dedication address (Wangensteen) 73 (Mar.)

Pineal gland
circadian rhythms and temperature variations in rats (Wiederanders et al.) 277 (Aug.)
relation to copper and calcium metabolism in rats (Wiederanders & Evans) 467 (Dec.)

Pregnancy
emotional problems, obstetrician's role in treatment (Bernstein) 325 (Sept.)
in patient with complete heart block treated with internal pacemaker (case report, Berestka & Spellacy) 461 (Dec.)
with osteogenesis imperfecta (case report, Dunham & Spellacy) 293 (Aug.)

Prostate gland
biopsy, perineal, disposable needle for (Mellinger) 103 (Mar.)
carcinoma, review of incidence, etiology, pathology (Blackard) 477 (Dec.)

Psychoneuroses
after leg amputation (Simpson & Albronda) 429 (Nov.)
determination of incompetency (O'Toole) 273 (Aug.)
in pregnancy, obstetrician's role in treatment (Bernstein) 325 (Sept.)
mental health characteristics of hospitalized college students (Koutsky) 397 (Oct.)

Psychotherapy, collaborative technique, Child Development Clinic, Children's Hospital of Winnipeg (Bell et al.) 68 (Feb.)

Q

Quadriplegia
mobility with, techniques and devices to aid patients (symposium, Hallin) 130 (Apr.)
traumatic, treatment and rehabilitation (case report, symposium, Martin) 112 (Apr.)

R

Radiation therapy, art and science 1965 (D'Angio) 256 (July)

Resuscitation in heart arrest, instruction by hospital anesthesia department (McCaughey) 46 (Feb.)

Rheumatic fever
acute, preventive measures and changes in diagnostic criteria and treatment (Lucas) 230 (July)
incidence in freshmen at University of North Dakota (Olmstead & Churchill) 317 (Sept.)

Roentgen examination
angiography for diagnosis of cerebral abscess with cyanotic congenital heart disease (case reports, Calkins & Bell) 403 (Oct.)
excretory urogram for diagnosis of vesicoureteral reflux (Creevy) 238 (July)
for diagnosis of renal cysts and tumors (Mellinger) 432 (Nov.)
for diagnosis of ruptured aneurysms of abdominal aorta (Nevin) 259 (July)
in carcinoma of prostate (Blackard) 477 (Dec.)
in primary lung cancer in elderly (Levine & Geller) 97 (Mar.)
oral changes in Wilson's disease (Ogle et al.) 464 (Dec.)

RUTH E. BOYNTON Series
Allopurinol in the treatment of gout (Seebach) 411 (Oct.)
Diagnosis of primary lung cancer in the aged patient (Levine & Geller) 97 (Mar.)

S

Seizures in children, classification and treatment (Copple & Isom) 167 (May)

Sex, transsexual research at the University of Minnesota (Hastings & Blum) 262 (July)

Speech
after correction of cleft palate (Waters) 60 (Feb.)
role of speech therapist in cleft palate treatment team (Vines) 62 (Feb.)

ST. PAUL–RAMSEY HOSPITAL SYMPOSIUM
Special Issue 229-261 (July)
Acute rheumatic fever: a new look (Lucas) 230
Chondritis as a postoperative complication (Lewis) 247
Introduction: A fitting tribute—a challenge (Peterson) 229
Neglected roentgen diagnostic feature of ruptured aneurysms of the abdominal aorta (Nevin) 259
Objective evaluation of auditory function (Long et al.) 251
Radiotherapy: art and science 1965 (D'Angio) 256
Vesicoureteral reflux: a brief review (Creevy) 238
What about the small bowel? (Gilchrist) 236

Surgery, yesterday and tomorrow: speech at Minneapolis Veterans Administration anniversary celebration (Wangensteen) 328 (Sept.)

Symposiums
Physical Medicine and Rehabilitation: part I, 109-142 (Apr.); part II, 187-209 (June)
St. Paul–Ramsey Hospital 229-261 (July)

T

Teeth deformities with cleft lip, palate, treatment (Cleall) 64 (Feb.)

Temperature, variations in normal, pincalectomized, and blinded rats (Wiederanders et al.) 277 (Aug.)

Thymus gland abnormality as cause of imminent death in infant (case report, Shannon) 78 (Mar.)

Thyroid gland, myxedema after removal of ectopic thyroid resembling thyroglossal duct cyst (case report, Saiki) 7 (Jan.)

Transfusions in factor VIII deficiency (Dana) 143 (May)

Travenol needle for perineal biopsy of prostate (Mellinger) 103 (Mar.)

Tumors
- hemangiopericytoma with pulmonary metastasis (case report, Bogedain & Carpathios) 213 (June)
- renal, differentiation from cysts (Mellinger) 432 (Nov.)

U

University of Minnesota
- early history of physical medicine and rehabilitation (symposium, Knapp) 136 (Apr.)
- history of physical medicine at, 1945 to 1965 (symposium, Olson) 203 (June)

University of Oregon Special Issue on Pediatrics 143-181 (May)

Urinary tract
- diagnosis of renal cyst and renal tumor (Mellinger) 432 (Nov.)
- disorders with myelopathies (symposium, Schutt et al.) 187 (June)
- incontinence in men, Berry prosthesis and silastic balloon for correction (Hoskins & Mellinger) 268 (July)

UROLOGY FOR THE GENERAL PRACTITIONER Series
- Carcinoma of the prostate: a review, Part I. Incidence, etiology, pathology, and diagnosis (Blackard) 477 (Dec.)
- Diagnosis of renal cyst versus renal tumor (Mellinger) 432 (Nov.)

V

Vitamins, C state, effect of alteration on carbohydrate metabolism (Setyaadmadja et al.) 18 (Jan.)

W

WANGENSTEEN Series see OWEN H. WANGENSTEEN Series

Wilson's disease, oral roentgenographic changes (Ogle et al.) 464 (Dec.)

XYZ

Zyloprim® (allopurinol) for gout (Seebach) 411 (Oct.)

AUTHOR INDEX

Acharya, V. N., Two-chambered right ventricle associated with primary pulmonary hypertension 210 (June)

Al-Attar, A. S., Pulmonary atresia in young adults 102 (Mar.)

Albronda, Henry F., Psychologic aspects of amputation of a lower limb 429 (Nov.)

Ashley, Carl G., Phenylketonuria: report of the Oregon detection and evaluation program 162 (May)

B

Babson, S. Gorham, Determination of gestational age in premature infants 174 (May)

Baird, D. A.
- An integrated approach to cleft lip and palate 55 (Feb.)
- Cleft lip 57 (Feb.)

Behrman, Richard E., Physiologic basis for the supportive management of the sick newborn 157 (May)

Bell, Anne, Child Development Clinic of The Children's Hospital and its method of collaborative treatment 68 (Feb.)

Bell, William E., Cerebral abscess and cyanotic congenital heart disease 403 (Oct.)

Berestka, S. A., Complete heart block associated with pregnancy and treated with an internal pacemaker 461 (Dec.)

Bernstein, Irving C., Functional problems confronting the obstetrician 325 (Sept.)

Bigourdan, Lillian, Strabismus: review of 100 surgical cases 49 (Feb.)

Bjorklund, Carl R., Physical treatment for the hemiplegic patient 123 (Apr.)

Blackard, Clyde E., Carcinoma of the prostate: a review: part I. Incidence, etiology, pathology, and diagnosis 477 (Dec.)

Blum, John A., A transsexual research project at the University of Minnesota Medical School 262 (July)

Bogedain, William, Therapy of hemangiopericytoma with pulmonary metastasis 213 (June)

Boies, L. R., Jr., The objective evaluation of auditory function 251 (July)

C

Calkins, Richard A., Cerebral abscess and cyanotic congenital heart disease 403 (Oct.)

Campbell, Daniel C., Patent ductus arteriosus: review of literature and experience with surgical corrections 415 (Oct.)

Campbell, Robert A., Combined immunosuppressive therapy in steroid-resistant, chronic glomerular diseases of childhood 149 (May)

Carpathios, John, Therapy of hemangiopericytoma with pulmonary metastasis 213 (June)

Chatikavanich, Kasarn, Oesophagopleural fistula: a complication of lobectomy, report of an unusual case 450 (Nov.)

Cheraskin, E., Ascorbic acid and carbohydrate metabolism: effect of supervised sucrose drinks upon two-hour postprandial blood glucose in terms of vitamin C state 18 (Jan.)

Chown, Bruce, The story of the Winnipeg Rh laboratory 38 (Feb.)

Christoferson, Lee A., Results from shunting procedures for internal hydrocephalus 321 (Sept.)

Churchill, Jane, Incidence of rheumatic fever: summary of an eight year study of incoming freshmen at the University of North Dakota 317 (Sept.)

Cleall, J.
An integrated approach to cleft lip and palate 55 (Feb.)
Dental aspects of the cleft lip and palate 64 (Feb.)

Cole, Theodore M., Osteoporosis 118 (Apr.)

Coodin, Fishel J., Some observations on Eskimo health 51 (Feb.)

Copple, Peggy J., Classification and treatment of seizure disorders in children 167 (May)

Creevy, C. D., Vesicoureteral reflux: a brief review 238 (July)

D

Dalessio, Donald J., Cerebral vasospasm in migraine 283 (Aug.)

Dana, George W., Transfusion therapy in factor VIII deficiency 143 (May)

D'Angio, G. J., Radiotherapy: art and science 1965 256 (July)

Dines, David E., Acute bronchiolitis as a cause of chronic obstructive lung disease in adults: report of two cases 281 (Aug.)

Dooley, Byron N., Patent ductus arteriosus: review of literature and experience with surgical corrections 415 (Oct.)

Dunham, Charles, Pregnancy in patients with osteogenesis imperfecta: a case report and a review of the literature 293 (Aug.)

E

Eckman, James, Biography of Frank H. Krusen, M.D. 140 (Apr.)

Engel, Rudolf, Electroencephalographic responses to sound and to light in premature and full term neonates 181 (May)

Evans, G. W.
Circadian rhythms and the pineal gland 277 (Aug.)
Copper and calcium metabolism in the rat: relationship to the pineal gland 467 (Dec.)

F

Farber, Seymour M., Development and perpetuation of obesity 13 (Jan.)

Fry, R., Atrial septal defects 385 (Oct.)

G

Geller, Steven, Diagnosis of primary lung cancer in the aged patient 97 (Mar.)

Geraci, Joseph E., Prolonged allergic reaction to oxacillin 10 (Jan.)

Gibilisco, Joseph A., Oral roentgenographic changes in Wilson's disease 464 (Dec.)

Gilchrist, Milton R., What about the small bowel? 236 (July)

Goldstein, Norman P., Oral roentgenographic changes in Wilson's disease 464 (Dec.)

Golwalla, A. F., Two-chambered right ventricle associated with primary pulmonary hypertension 210 (June)

Grant, W. Wallace
Child Development Clinic of The Children's Hospital and its method of collaborative treatment 68 (Feb.)
Introduction, Special Issue on Pediatrics, Children's Hospital of Winnipeg 33 (Feb.)

Gregware, P. R., Atrial septal defects 385 (Oct.)

Guenter, Clarence A., Acute myocardial infarction complicated by complete heart block: use of the transvenous pacemaker 393 (Oct.)

Gustafson, Arthur A., Contagious ecthyma in man: report of two cases with laboratory studies 400 (Oct.)

H

Hallin, Roger P., Transfers and mobility for paraplegic and quadriplegic patients 130 (Apr.)

Halpern, Daniel, Physical therapy in obstructive pulmonary disease 125 (Apr.)

Hamilton, C. A., Atrial septal defects 385 (Oct.)

Hanada, M., Effect of LSD on the phantom limb phenomenon 22 (Jan.)

Hastings, Donald W., A transsexual research project at the University of Minnesota Medical School 262 (July)

Haworth, J. C., Neonatal hypoglycemia: a six-year experience 41 (Feb.)

Henderson, Edward D., Prolonged allergic reaction to oxacillin 10 (Jan.)

Hilger, Jerome A.
biography of Henry L. Williams, Jr., M.D. 485 (Dec.)
Objective evaluation of auditory function 251 (July)

Hood, Richard H., Jr., Patent ductus arteriosus: review of literature and experience with surgical corrections 415 (Oct.)

Hoskins, John H., A promising procedure for the treatment of urinary incontinence in the male 268 (July)

Hunt, Carl E., Eosinophilic peritonitis: report of two cases 473 (Dec.)

I

Isom, John B., Classification and treatment of seizure disorders in children 167 (May)

J

Jacinto, Elsa Y., Combined immunosuppressive therapy in steroid-resistant, chronic glomerular diseases of childhood 149 (May)

Johanson, Waldemar G., Acute myocardial infarction complicated by complete heart block: use of the transvenous pacemaker 393 (Oct.)

K

Kasahara, Y., Effect of LSD on the phantom limb phenomenon 22 (Jan.)

Keith, Dorothy, Child Development Clinic of The Children's Hospital and its method of collaborative treatment 68 (Feb.)

Kernahan, D. A., An integrated approach to cleft lip and palate 55 (Feb.)

Kimbrough, L. S., Development and perpetuation of obesity 13 (Jan.)

Kinare, S. G., Two-chambered right ventricle associated with primary pulmonary hypertension 210 (June)

Knapp, Miland E.
The early history of physical medicine and rehabilitation at the University of Minnesota 136 (Apr.)
The relationship of the staff physician to the department of physical medicine and rehabilitation of a community hospital 207 (June)

Konsuwan, Nit, Oesophagopleural fistula: a complication of lobectomy, report of an unusual case 450 (Nov.)

Kottke, Frederic J.
Physical medicine and rehabilitation as a medical specialty 109 (Apr.)
Physiology of balance 197 (June)

Koutsky, Carl D., Psychiatric hospitalization of students on a college campus 397 (Oct.)

Krivit, William, Eosinophilic peritonitis: report of two cases 473 (Dec.)

Kuromaru, S., Effect of LSD on the phantom limb phenomenon 22 (Jan.)

L

Lambie, John A., Contagious ecthyma in man: report of two cases with laboratory studies 400 (Oct.)

Lees, Martin H., Heart failure in infancy 178 (May)

Levine, Ida, Diagnosis of primary lung cancer in the aged patient 97 (Mar.)

Lewis, F. John, Chondritis as a postoperative complication 247 (July)

Lind, K. A., Circadian rhythms and the pineal gland 277 (Aug.)

Long, Sister Victorine, The objective evaluation of auditory function 251 (July)

Lowe, Sandra, Child Development Clinic of The Children's Hospital and its method of collaborative treatment 68 (Feb.)

Lucas, Russell V., Jr., Acute rheumatic fever: a new look 230 (July)

Lundy, John S.
Dr. Ralph Moore Tovell 291 (Aug.)
The mystery of atypical pain 283 (Aug.)

M

Martin, Gordon M.
Perceptual problems of the hemiplegic patient 193 (June)
Practical rehabilitation 112 (Apr.)
Problems of the bladder and bowel in patients with myelopathies 187 (June)

Martin, William J.
Bacteremia: common pathogens and methods of management 439 (Nov.)
The penicillins 79 (Mar.)

McCaughey, T. J., Teaching resuscitation in a children's hospital 46 (Feb.)

McKinnon, C. M., Determination of gestational age in premature infants 174 (May)

McRae, K. N.
Child Development Clinic of The Children's Hospital and its method of collaborative treatment 68 (Feb.)
Neonatal hypoglycemia: a six-year experience 41 (Feb.)

Medovy, Harry, Pediatrics in Manitoba: goals and accomplishments 34 (Feb.)

Mellinger, George T.
A promising procedure for the treatment of urinary incontinence in the male 268 (July)
Diagnosis of renal cyst versus renal tumor 432 (Nov.)
Perineal biopsy of the prostate with a new disposable biopsy needle 103 (Mar.)

Michaelsen, G. S., Institutional environmental health and safety: the Minnesota story 1 (Jan.)

Myers, J. Arthur, biography of Owen H. Wangensteen, M.D. 216 (June)

N

Narang, R. K., Topical pulmonary eosinophilia: a clinical study 457 (Nov.)

Nelson, Edward N., Eosinophilic peritonitis: report of two cases 473 (Dec.)

Neville, William E., Mediastinal abscess of subdiaphragmatic origin 265 (July)

Nevin, I. Nik, A neglected roentgen diagnostic feature of ruptured aneurysms of the abdominal aorta 259 (July)

Nickerson, Warren R., Mediastinal abscess of subdiaphragmatic origin 265 (July)

O

Ogle, Richard G., Oral roentgenographic changes in Wilson's disease 464 (Dec.)

Okada, S., effect of LSD on the phantom limb phenomenon 22 (Jan.)

Olin, Ronald, Diseases of medical progress 390 (Oct.)

Olmstead, Edwin G., Incidence of rheumatic fever: summary of an eight year study of incoming freshmen at the University of North Dakota 317 (Sept.)

Olmsted, Richard W., Prefatory remarks: Special Issue on Pediatrics, University of Oregon 143 (May)

Olson, Mildred E., History of physical medicine at the University of Minnesota from 1945 to 1965 203 (June)

Opstad, Earl T., biography of Karl W. Anderson, M.D. 421 (Oct.)

O'Toole, James K., Incompetency 273 (Aug.)

Owens, Guy, Control of pain in patients with cancer 287 (Aug.)

P

Papermaster, Theodore C., Eosinophilic peritonitis: report of two cases 473 (Dec.)

Peters, Gustavus A., Prolonged allergic reaction to oxacillin 10 (Jan.)

Peterson, D. H., A fitting tribute—a challenge 229 (July)

Pool, Thomas L., Problems of the bladder and bowel in patients with myelopathies 187 (June)

R

Ramos, P., Atrial septal defects 385 (Oct.)

Randall, Raymond V., Oral roentgenographic changes in Wilson's disease 464 (Dec.)

Reed, Howard, Strabismus: review of 100 surgical cases 49 (Feb.)

Ringsdorf, W. M., Jr., Ascorbic acid and carbohydrate metabolism: effect of supervised sucrose drinks upon two-hour postprandial blood glucose in terms of vitamin C state 18 (Jan.)

Rossi, Nicholas P., Heart-lung bypass: perspective and present status 89 (Mar.)

Roth, N. A., The objective evaluation of auditory function 251 (July)

S

Saiki, John H., Severe myxedema following inadvertent removal of an ectopic thyroid resembling a thyroglossal duct cyst 7 (Jan.)

Sakamoto, K., Effect of LSD on the phantom limb phenomenon 22 (Jan.)

Sawtell, Robert R., Perceptual problems of the hemiplegic patient 193 (June)

Schutt, Ann H., Problems of the bladder and bowel in patients with myelopathies 187 (June)

Seebach, Lydia M., Allopurinol in the treatment of gout 411 (Oct.)

Setyaadmadja, A.T.S.H., Ascorbic acid and carbohydrate metabolism: effect of supervised sucrose drinks upon two-hour postprandial blood glucose in terms of vitamin C state 18 (Jan.)

Shannon, W. Ray, Episode of imminent death in a three-week-old infant 78 (Mar.)

Shapiro, Sidney K., biography of A. B. Baker, M.D. 105 (Mar.)

Simpson, Elaine B., Psychologic aspects of amputation of a lower limb 429 (Nov.)

Spellacy, W. N.

Complete heart block associated with pregnancy and treated with an internal pacemaker 461 (Dec.)

Pregnancy in patients with osteogenesis imperfecta: a case report and a review of the literature 293 (Aug.)

Stewart, Janet M., Phenylketonuria: report of the Oregon detection and evaluation program 162 (May)

Stillwell, Dorothy M., Physical medicine for patients with rheumatic disease 133 (Apr.)

Store, S. D., Two-chambered right ventricle associated with primary pulmonary hypertension 210 (June)

T

Thomson, Stewart C., biography of Gaylord W. Anderson, M.D. 30 (Jan.)

V

Vines, T.

An integrated approach to cleft lip and palate 55 (Feb.)

Role of the speech therapist on the cleft palate team 62 (Feb.)

W

Wangensteen, Owen H.

Surgery—yesterday and tomorrow 328 (Sept.)

Surgical education and the training of surgeons 73 (Mar.)

Wannamaker, Lewis W., biography of John A. Anderson, M.D. 270 (July)

Waters, W. R.

An integrated approach to cleft lip and palate 55 (Feb.)

Treatment of cleft palate 60 (Feb.)

Way, R. Clifton, Heart failure in infancy 178 (May)

Weaver, Floyd E., Mediastinal abscess of subdiaphragmatic origin 265 (July)

Whang, Robert, Hyperglycemic nonketotic coma induced by peritoneal dialysis 453 (Nov.)

Wiederanders, R. E.

Circadian rhythms and the pineal gland 277 (Aug.)

Copper and calcium metabolism in the rat: relationship to the pineal gland 467 (Dec.)

Wilson, Nancy L., Development and perpetuation of obesity 13 (Jan.)

Wilson, Roger H. L., Development and perpetuation of obesity 13 (Jan.)

XYZ

Zhdanov, Viktor M., The 1967 influenza B in the USSR 419 (Oct.)

Letters to the Editor . . .

Studies Done at St. Paul–Ramsey

The article entitled "Acute Myocardial Infarction Complicated by Complete Heart Block: Use of the Transvenous Pacemaker with Observations on a New Demand Pacemaker" which appeared in the October 1967 issue of your journal did not recognize the St. Paul–Ramsey Hospital as the site of the studies.

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Asst. Prof. Medicine and Physiology
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LSD Influence

Since publishing my article entitled, "Hospitalization of Students on a College Campus," in the October issue of "JOURNAL-LANCET," there have been other factors brought to bear on the student body that need comment.

As of January 1965, the facts and figures are true representation of hospitalization of students in the psychiatric service. However, shortly following this cutoff date, we did begin to have admitted to our hospital, psychotic reactions that were related to the use of LSD by the student body. The number was not very large and I am uncertain as to how many more of these instances have occurred since I have left the University service to practice in Anchorage, Alaska. However, I think the number of students admitted significantly reflected the seriousness of the LSD influence in certain cases.

CARL D. KOUTSKY, M.D.
Superintendent, Alaska Psychiatric Institute
Anchorage

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News Briefs . . .

North Dakota

ELECTED American College of Surgeons governor for North Dakota at the organization's convention in Chicago was Dr. R. F. Nuessle of the Quain and Ramstad Clinic, Bismarck. Inducted into the College were Drs. Norman Ordahl, Dickinson, and Hugh D. Thorfinnson and Richard H. Seifert, Fargo.

* * * *

CERTIFIED in their medical specialties were 2 physicians of the Neurologic Associates of Fargo: Dr. Richard A. Olafson, who completed his requirements for the American Board of Neurological Surgery, and Dr. Robert R. Ivers, qualified by the American Board of Psychiatry and Neurology.

* * * *

DR. ROGER D. ENGBERG, formerly associated with the DePuy-Sorkness Clinic of Jamestown, moved his office to the Orthopedic Clinic Building in that city.

* * * *

DR. WILLIAM D. ERICKSON, a native of Jamestown, joined the staff of the Children's Clinic there.

* * * *

DR. ROBERT L. JENNINGS of Bismarck was elected to partnership in the Capital City Clinic.

DR. GORDON MAGILL of Fargo was elected to a third term as president of the Cass County unit of the American Cancer Society.

* * * *

DR. T. H. HARWOOD, dean of the University of North Dakota School of Medicine, was featured speaker at the annual meeting of the North Dakota division of the American Cancer Society at Jamestown.

* * * *

DR. C. H. PETERS of Bismarck was named president-elect of the North Dakota Conference of Social Welfare.

* * * *

DR. KENNETH S. HELENBOLT of Grand Forks is the new president of the North Dakota chapter of the American College of Surgeons. Other officers are Drs. Henry Norum of Fargo, vice-president, and Grandon E. Tolstedt of Bismarck, secretary-treasurer.

* * * *

AN OFFICE was opened in Grand Forks by the North Dakota Regional Medical Program, organized to review and facilitate the care of heart disease, cancer, and stroke patients in the state. Dr. T. H. Harwood, dean of the University of North Dakota School of Medicine, is coordinator of the program, established by the North Dakota Medical Research Foundation. Named to the

(Continued on page 12A)

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FREE PARKING

(Continued from page 10A)

executive committee of the program were Drs. L. A. Christoferson, Fargo, chairman; R. C. Painter, Grand Forks, vice-chairman; J. R. Amos, Bismarck, secretary; A. Malcolm Cameron, Minot; J. D. Craven, Williston; P. Roy Gregware, Bismarck; E. O. Hieb, Jamestown; D. A. Nicely, Dickinson; J. T. Goven, Valley City; and R. D. Story and R. Ulmer, Fargo.

Minnesota

DR. H. L. WILLIAMS, emeritus staff member and former head of the section of otolaryngology and rhinology of the Mayo Clinic, Rochester, was presented with a commemorative plaque during an international symposium on Meniere's disease, at which he was the guest of honor, at Rochester. Dr. Williams is now in charge of graduate medical training in diseases of the ear, nose, and throat at Veterans Administration Hospital, Minneapolis.

* * * *

DR. GARY R. FEIGAL, a native of Pine Island, joined Drs. David Sontag and E. C. Bayley in practice at Lake City.

* * * *

DRS. HERMAN J. JUST and JOHN FODERICK of Hastings dissolved their professional association.

* * * *

DR. ALAEDDIN MOGHADDAM, a native of France, joined the staff of the St. Louis Park Medical Center.

* * * *

DR. C. E. RHODES of the Park Rapids Clinic was elected to active membership in the American Academy of General Practice.

* * * *

DR. DONN R. DRIVER, former chief of medical services at the Veterans Administration Center, Sioux Falls, S.D., was appointed chief of the outpatient service at Veterans Administration Hospital, Minneapolis.

* * * *

DR. WILSON M. SALTER, a native of Virginia, joined the Willmar Clinic as a specialist in internal medicine.

* * * *

DR. J. A. COSGRIFF, JR., of Olivia attended the annual meeting of the Association of American Physicians and Surgeons in Houston, Texas, and presented the address which was to have been given by his father, the late Dr. J. A. Cosgriff, Sr., national president of the organization.

* * * *

APPOINTED by the Mayo Clinic, Rochester, as a consultant in diagnostic roentgenology was Dr. Glen Hartman. Six new associate consultants named were Drs. George Beeler, Jr., physiology; Gerald Gau and John Merideth, cardiology; Stanley Payne, orthopedic surgery; Charles Rife, urology; and Fenwick Riley, Jr., ophthalmology. Drs. Norman Odyniec and Paul Roy were appointed chief resident associates in surgery.

* * * *

DR. EARL CARTER of the Mayo Clinic, Rochester, participated in a conference on aerospace medicine at Ohio State University, Columbus.

* * * *

DR. MILTON ADLER, associate professor of neurology at the University of Minnesota, was appointed chief of neurology at Veterans Administration Hospital, Minneapolis.

(Continued on page 14A)

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NEWS BRIEFS

(Continued from page 12A)

DR. AUSTIN M. MCCARTHY, Willmar Clinic surgeon, addressed a North Dakota chapter of the American College of Surgeons meeting on hip fractures and the use of metallic prostheses in such fractures.

* * * *

DR. A. B. BAKER, University of Minnesota neurology division chief, received a $385,010 grant from the Public Health Service of the Department of Health, Education, and Welfare for the seventh year of a twelve-year neurological research project on strokes.

* * * *

DR. JO E. ANDERSON of Le Sueur was a guest lecturer at the Fourth Annual Hospital Medical Staff Conference sponsored by the University of Colorado at Denver.

* * * *

DR. LYNN C. STOKER of Albert Lea became a fellow of the American College of Surgeons.

* * * *

DR. DONALD ERICKSON of the Mayo Clinic, Rochester, spoke on the "Use of Physical Modalities" at the Ninth National Conference on the Medical Aspects of Sports at Houston, Texas.

* * * *

DR. T. R. FRITSCHE of New Ulm received the Harold S. Diehl Outstanding Physician Award from the University of Minnesota Medical Alumni Association.

* * * *

DR. E. P. K. FENGER, Minnesota director of tuberculosis services, was awarded the Hoyt E. Dearholt Medal of the Mississippi Valley Conference on Tuberculosis in recognition of his outstanding work in tuberculosis and other pulmonary diseases.

* * * *

DR. DONALD PETERSON, director of the alcohol and drug addiction unit at Willmar State Hospital and chairman of the Minnesota Medical Association's subcommittee on alcoholism, was appointed by Gov. Harold LeVander to the Minnesota Commission on Alcohol Problems.

* * * *

DR. JOHN HIGGINS of Rochester was elected secretary-treasurer of the Alumni Association of Mayo Graduate School.

* * * *

PRESENTING PAPERS or participating in panel discussions at the annual meeting of the American Academy of Pediatrics at Washington, D. C., were Drs. Thane Cody, Gunnar Stickler, and Hugh Lynn of the Mayo Clinic, Rochester, and Dr. Thomas Tachovsky, a resident in the Mayo Graduate School of Medicine.

* * * *

DR. FRANCIS TYCE, medical director of Rochester State Hospital, was installed as president of the Association of Medical Superintendents of Mental Hospitals at the national organization's semiannual convention in Minneapolis.

* * * *

DR. V. P. GEORGE of Le Sueur was reelected to the board of directors of the Minnesota division of the American Cancer Society.

* * * *

DR. D. J. DUMMER of the New Brighton Clinic was reelected to active membership in the American Academy of General Practice.

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PROMOTED to professor in the Mayo Graduate School of Medicine were the following Mayo Clinic, Rochester, physicians: Drs. Norman Goldstein, neurology; Richard Theye, anesthesiology; George Baker, clinical neurosurgery; John Hodgson, clinical radiology; and Lloyd Bartholomew, Edwin Bayrd, and William Summerskill, medicine.

* * * *

NEW OFFICERS of the Northwestern Pediatric Society include Dr. Lloyd E. Harris, Mayo Clinic, Rochester, president-elect, and Dr. Stanley A. Leonard, St. Paul, secretary-treasurer.

* * * *

DR. E. P. BRYANT closed his practice in the Ellendale Clinic to go into group practice at Silver Bay.

* * * *

DR. WILLIAM C. LEE, anesthesiologist at Hibbing General Hospital, was granted a fellowship in the American College of Anesthesiologists.

* * * *

DR. R. A. MACDONALD of Grand Marais received the American Cancer Society's National Divisional Award in recognition of his uterine cancer detection work in Cook County.

* * * *

DR. R. G. HANKERSON, formerly of Minnesota Lake, joined the Mankato State College health service.

* * * *

HONORED by the American Roentgen Ray Society were Dr. John Kirklin, former Mayo Clinic heart surgeon, who received the Caldwell Medal, the Society's highest award, and Dr. Allen Good, head of the Mayo Clinic section of diagnostic X-ray, who was installed as president.

* * * *

DR. HARRY BISEL, head of the Mayo Clinic clinical oncology section, was elected to the board of directors of the Minnesota division of the American Cancer Society.

* * * *

DR. EDWARD H. SOULE of the section of surgical pathology of the Mayo Clinic, Rochester, and associate professor of pathology in the Mayo Graduate School of Medicine, was awarded the gold medal, given for the best educational exhibit of the American Society of Clinical Pathologists, at the organization's annual meeting in Chicago. An earlier version of the exhibit, "Primary Tumors of the Soft Tissues of the Extremities," won a certificate of merit at the 1967 meeting of the American Medical Association.

South Dakota

INDUCTED at Chicago into membership in the American College of Surgeons were Drs. William A. Bormes, Aberdeen; Russell H. Harris, Rapid City; and H. Phil Gross, Sioux Falls.

* * * *

DR. E. G. HUPPLER of Watertown was elected president of the South Dakota division of the American Cancer Society.

(Continued on page 16A)

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References:
1. Cuomo, F. C. and Cantarjes, O.: *New York State J. Med.* 52: [illegible] (Mar. 15) 1952.
2. Xanders, C. W.: *Western Med.* 1: 14 (Sept.) 1960.
3. Baker, A. G.: *Prairie Med. J.* 63: 697-698 (May) 1960.
4. Lehrer, H. W., Lehrer, H. G. and Lehrer, D. R.: *Northwest Med.* 75: 1249-1252 (Nov.) 1955.

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(Continued from page 15A)

DR. E. T. LIETZKE of Beresford attended the annual meeting of the American Academy of General Practice in Dallas, Texas. He is a member of the house of delegates of the academy, which consists of 2 members from each of the 50 states, the District of Columbia, and Puerto Rico.

Deaths . . .

DR. THEODORE HIGGINS SWEETSER, SR., 74, retired chief of urology at 2 Minneapolis, Minn., hospitals and a pioneer in correcting bladder defects in infants, died Oct. 8. He had headed the urology divisions of St. Mary's and Hennepin County General hospitals from the late 1930s until his retirement several years ago. His surgical technique to correct exstrophy has been adopted by hospitals in other parts of the country and abroad.

* * * *

DR. J. E. STUDENBERG, 60, Winner, S.D., died Oct. 9. He practiced in Midland, Philip, and Gregory, S.D., and saw duty in the Pacific in World War II before coming to Winner in 1945.

DR. IAN DAVIS TIEDEMANN, 77, formerly of Heron Lake, Minn., died in Whittier, Calif., Oct. 10. A native of West Concord, Minn., Dr. Tiedemann owned the hospital in Heron Lake before moving to California in 1927.

* * * *

DR. ALLAN A. BAILEY, 57, former head of a section of neurology at the Mayo Clinic, died in Rochester, Minn., Oct. 3. A native of Canada, Dr. Bailey was chief of the department of medicine at the University of Saskatchewan, Saskatoon, at the time of his death.

* * * *

DR. WILLIAM E. PROFITT, JR., 52, Minneapolis, Minn., died Sept. 30. An outstanding athlete at the University of Minnesota in the 1930s, he had been the Minnesota Twins team physician for the past seven years. He also was chairman of the Governor's Council on Youth Physical Fitness.

* * * *

SIR JAMES LEARMONTH, 72, a former neurologic surgeon at the Mayo Clinic, Rochester, Minn., died in his native Scotland, Sept. 27. A member of the Clinic staff from 1928 to 1932, Sir James performed surgery to restore the circulation in the leg of King George VI of England in 1949.

* * * *

DR. ERNEST M. HAMMES, 83, a University of Minnesota professor emeritus of neurology and psychiatry, died in Minneapolis, Minn., Sept. 30. He was a past president of the Ramsey County Medical Society, the Minnesota State Medical Association, and Blue Cross of Minnesota, which he helped organize.

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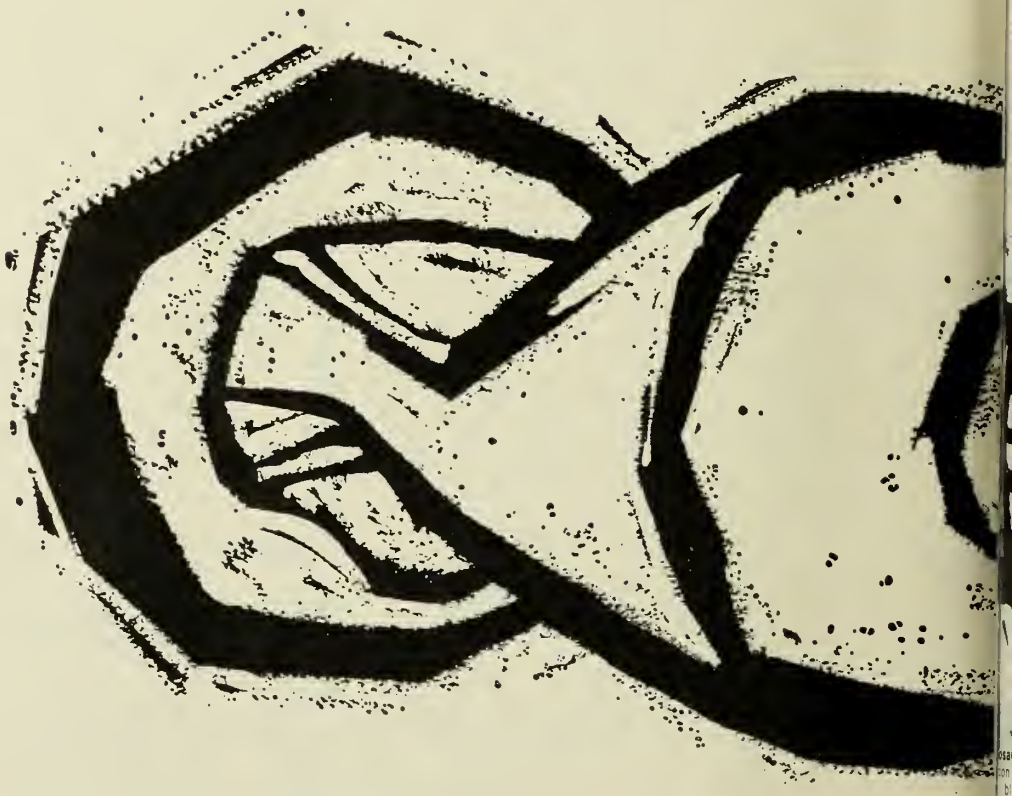
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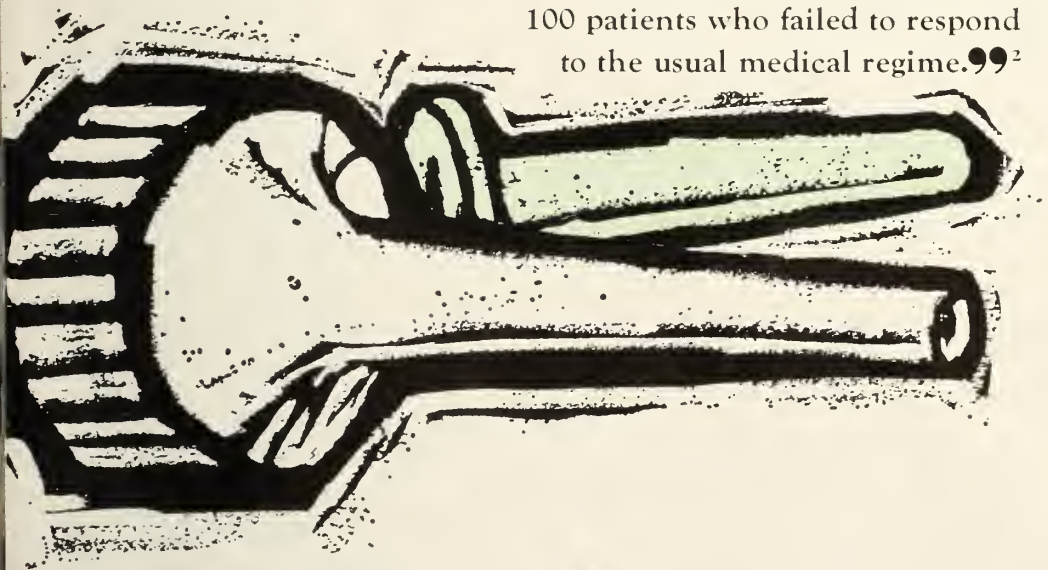
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"...continued good results in more than 100 patients who failed to respond to the usual medical regime."[2]



sage and Administration: Use of Cortenema is predicated on concomitant use of modern supportive measures, such blood replacement, rational dietary control, antispasdics and sedatives, antibacterial therapy, and systemic roids.

Usual dose is one Cortenema daily for 2 or 3 weeks and ery second day thereafter, administered intrarectally in ening before retiring. Every effort should be made to re- n medication for at least one hour, preferably all night. is may be facilitated by prior sedation and/or anti- rrheal medication. If improvement does not occur within or three weeks, discontinue use.

nstruct patient to lie on left side during instillation. ake bottle vigorously to resuspend hydrocortisone. Exse lubricated tip by removal of sheath. *Carefully* insert into rectum in direction of sacrum. Slowly express con- ts by compressing container. After instillation, patient uld remain on left side at least 30 minutes.

Duration of treatment depends on degree of response. nptomatic response usually occurs within 5 to 7 days. provement in mucosa, gauged by barium enemas and moidoscopic examinations, may lag somewhat behind ical improvement. Usual duration is two weeks, although ne cases require up to 2 or 3 months. Minimal control symptoms is insufficient basis for prolonged use. If no ical or proctologic response occurs within 2 or 3 weeks, if patient's condition worsens, discontinue use.

ymptomatic improvement should not be sole criterion udging efficacy. Actual sigmoidoscopic examination and ay visualization are most reliable. Since steroids can bit wound healing, Cortenema should not be used in ediate or early postoperative period following ileorecomy.

v Supplied: In boxes of 7 single-dose units.

tion: Federal law prohibits dispensing without a preption.



For additional information, reprints or sample, write

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1. Sherman, L. F.: The treatment of ulcerative colitis with hydrocortisone alcohol retention enemas. A topical and systemic anti-inflammatory agent, *Minn. Med.* 48:605, May 1965.

2. Sherman, L. F., Tenner, R. J. and Schottler, J. L.: Treatment of ulcerative colitis with hydrocortisone alcohol retention enemas: A topical and systemic anti-inflammatory agent. Part II, *Dis. Colon & Rectum* 10:43, January-February 1967.



Help her regain equanimity

Mebaral®
brand of
mephobarbital

TABLETS 32 mg. (½ grain)
50 mg. (¾ grain)
100 mg. (1½ grains)

For the anxious patient who must think clearly all day long, Mebaral usually provides dependable sedation without the degree of languor or decrease in alertness caused by other barbiturates.[1]

Mebaral is of value in the treatment of anxiety-tension states[2] when minimal hypnotic action is desired. Its sedative action is prolonged[3] and predictable.

Dosage: *Adults,* for daytime sedation—½ grain (32 mg), ¾ grain (50 mg.) and, at times, 1½ grains (100 mg.), three or four times daily.

Side effects: Although Mebaral is generally well tolerated over long periods, the possibility of idiosyncrasy to barbiturates (as manifested by drowsiness, vertigo and cutaneous eruptions) should be considered. As with other barbiturates, caution is advisable during use in debilitated and senile patients and in patients with pulmonary disease. Large doses are contraindicated in patients with nephritis.

Dependable daytime sedation

References: 1. Musser, Ruth D., and Shubkagel, Betty L.: Pharmacology and Therapeutics, ed. 3, New York, Macmillan Company, 1965, p. 363. **2.** Council on Drugs, American Medical Association: New Drugs 1965, Chicago, American Medical Association, 1965, p. 157. **3.** Modell, Walter (Ed.): Drugs in Current Use 1966, New York, Springer Publishing Company, Inc., 1966, p. 77.

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